DIAGNOSIS OF OCCUPATIONAL DISEASES

Editors

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PREFACE

For more than a decade PRIA has been involved in the issues of workplace health and safety. Although workers and their organisations have been our primary constituency, we also work with doctors and other experts on the subject. Not only in India but also in other developing countries, occupational diseases are mostly improperly diagnosed. One of the reason is the absence of proper guidelines for diagnosis. The objective of this publication is not only to fill this gap but also contribute to the overall movement of providing justice to millions of workers. We have tried to keep the document as simple as possible and provide latest information on the subject.

`Diseases at work' was an admirable publication of PRIA in 1988. It contained detailed yet concise and compact information to diagnose occupational diseases of schedule III of the Workmen's Compensation Act (1923) and Employees State Insurance Act (1948). It was also in a language which non-doctors could understand.

In mid 1995 it was decided to review "Diseases at Work" and bring out a document mainly for doctors in government and non-government occupational health clinics and other related set-ups. The document had to be updated with recent reports published on the subject and also made concise and compact. Two background papers prepared by the doctors in Mumbai and Calcutta were circulated among doctors and experts in the field of Occupational Health all over India. The papers were written based on extensive surveys of the published and some non-published materials.

After getting comments from eminent doctors and others working in the field, a meeting was called in Delhi in June, 1996, to discuss each of the diseases in detail. The comments of the meeting were incorporated and a new document was recirculated for comments to doctors all over the country, which included eminent doctors

from major teaching hospitals of all the four metropolitan cities viz.: New Delhi, Bombay, Madras and Calcutta.

The document is to be given to all medical colleges, government hospitals and all clinics dealing with occupational health. It is hoped that doctors would get an opportunity to read this book and hopefully change the presently depressing scenario of occupational health in India. A training programme for doctors on the subject would be undertaken in various parts of India within a short period of time.

Along with the team of the Centre for Occupational and Environmental Health (PRIA), I am thankful to Mr.Vijay Kanhere and Dr. V. Murlidhar for compiling this document. We are also grateful to all the medical experts who contributed by their valuable comments in preparing this document.

Dr. Rajesh Tandon
Executive Director
Society for Participatory Research in Asia (PRIA)
New Delhi

INTRODUCTION

`Patient presented with the following symptoms', many a diagnosis begins thus, but very few end in `The patient suffers from occupational disease'.

Only few cases of occupational dermatitis were recorded officially in whole of India in the 80's. This is the situation of all recorded incidences of occupational diseases as most of them are not recorded at all.

That is the reason why official statistics does not reflect the ground reality. The conditions in mills, chemical units, pharmaceuticals, engineering companies, mines, in home based work, in agriculture and even in modern computer rooms are not hygienic. Studies have shown that at least forty percent of workers in spinning sections of textiles, are affected due to byssinosis, an occupational lung disease caused by cotton dust. The number of patients of byssinosis if properly recorded will be in thousands in one city of Mumbai alone.

There are many reasons for the fact that many patients are not being recognised as being affected due to occupational diseases. One is that they are not diagnosed correctly. One of the reasons for this is even if a doctor is interested and keen, he/she is not trained properly in diagnosis of occupational diseases.

In the undergraduate course of medicine little attention is paid to occupational diseases. Sometimes they are covered briefly under, `Preventive and Social Medicine'. Only few courses are available in India for post graduation studies in the field of occupational health. That is the reason that only few trained doctors are available to treat workers who are exposed to hazardous working environment.

The extent of neglect in this field becomes obvious when we contrast the pathetic recording of the occupational cancers and skin diseases to the fact that two thirds of carcinogens were first

identified in occupational setting and that occupational skin diseases are rampant.

We decided to fill the lacunae in a small way by putting together these guidelines on diagnosis. There are many occupational diseases but we have restricted ourselves to the official legal list of occupational diseases in schedule III of the Employees State Insurance Act 1948 and the Workmen's Compensation Act 1923.

These guidelines obviously cannot be a substitute for reference books/volumes. We have attempted to put together a set of symptoms which are observed and listed in reference books. As a useful but not exhaustive indicator we have also put together the possible industrial occurrences.

Biological monitoring in case of some substances is also added.

One question is asked to us time and again, `Why do you begin from substances causing diseases? Why not, as usual, begin from symptoms?'. When arriving at a diagnosis of a case, `Who is the patient?' is recognised as an important aspect of the history: Old or young , man or woman, child or grown up; where does the patient live?. These are traditionally accepted as important pointers towards causation of the disease.

There is a need to broaden history-taking to compulsorily include occupational history which may be a crucial factor in the causation of disease.

For example, dry cell factory workers may be exposed to manganese. They may present with psychological problems. These may be due to exposure to manganese associated with other stresses. If occupational origin is not elicited at an early stage the treatment may go haywire.

This is all the time happening in case of dust related lung diseases. TB is a handy diagnosis in such cases. If occupational history were to be recorded, then many a silicotic patients would have been saved from exposure to TB patients in TB hospitals.

Prevention needs no emphasis. For example, if workers exposed to benzene have to wait till the symptoms appear (leukemia, aplasticanaemia) it may be too late. If occupational history were recorded and interpreted properly many a disease could be prevented.

You may use the index of symptoms and body parts affected or the list of substances causing well known symptoms such as lung irritation. One may even begin from the index of industrial occurrence.

We have tried to make this book as useful as possible. Initially Society for Participatory Research in Asia (PRIA), Delhi had published `reference sheets on Notifiable Diseases'. These were about occupational diseases listed in the Factories Act, 1948.

In 1989 this was followed by a set of reference sheets on diseases in schedule III of the Workmen's Compensation Act, 1923, (Diseases at work II). These were in form and language easier for worker activists to understand.

In 1996 DISHA (Calcutta) with PRIA put together a similar material and held a meeting of doctors about the above. Dr. Murlidhar V, Mumbai presented a set of symptoms relating to many hazardous substances.

Eminent doctors discussed the above three documents relating to schedule III of Workmen's Compensation Act (1923) and Employees' State Insurance Act (1948) and gave suggestions in the course of three days. Later Dr. N.K. Mehrotra (Deputy Director ITRC Lucknow), Dr. S. K. Sharma (AIIMS, Delhi), Dr. H.N. Saiyed (RIOH,Calcutta), gave many important suggestions. Dr. Mehrotra had been very supportive continuously from the first publication on Notifiable Diseases in 1987.

We are thankful to Mr. Harsh Jaitli for coordinating the national level meetings and discussions from time to time. We also thank Sumedha Saxena for helping in the editing of this book. Thanks

are also due to the rest of PRIA team who have helped in various capacities.

We have edited this version in this form and are responsible for the lacunae in this publication. We will appreciate all suggestions and comments and criticisms about this book.

Vijay Kanhere
Dr. Murlidhar V.

LIST OF SPECIALISTS WHO COMMENTED ON THE PRELIMINARY DOCUMENT AT THE DELHI MEETING AND LATER ON BY CORRESPONDENCE

Dr. J. Amalorpavanathan	Assistant Professor Surgery, Madras Medical College
Dr. B.S. Chouhan	Desert Science Centre, Jodhpur
Dr. Kunal Datta	Medical Superintendent ESI Hospital, Belur, Calcutta
Mr. Vijay Kanhere	Occupational Health and Safety Centre (OHSC), Mumbai and PRIA, New Delhi
Dr. Swapan Kumar Das	21 Lal Goch , Calcutta
Dr. Abhay Shukla	992/993-23, Rajendranagar, Pune
Dr. Murlidhar V.	Associate Professor Surgery, LTM Medical College, Mumbai; Consultant OHSC, Mumbai
Dr. Veena Murlidhar	Occupational Health and Safety Centre (OHSC), Mumbai
Dr. Sunil Ghate	Department of Skin and STD, L.T.M.G. Hospital and LTM Medical College, Mumbai
Dr. Prathibha Gupta	Professor of Physiology, University College Medical Sciences, New Delhi
Dr. P.K. Ganguly	Regional Occupational Health Centre, Calcutta
Dr. N.K. Mehrotra	Deputy Director, Industrial Toxicology Research Centre (ITRC), Lucknow
Mr. Harsh Jaitli	Occupational Health Expert, PRIA, New Delhi
Dr. S.K. Sharma	Associate Professor, AIIMS, New Delhi
Dr. H.N. Saiyed	Regional Occupational Health Centre (E), Calcutta.

SIGNIFICANCE OF SCHEDULE III

When any worker is affected due to any disease listed in schedule III the important factor is to note the occupational risk involved. Occupational history and presence of the diseases listed are two most important aspects. Law presumes the interconnection between Occupational exposure and disease in case of Schedule III. If there is any contention of any other cause than occupational exposure, then that contention has to be supported by separate facts. For the working person, it is only needed to prove that the risk is involved in the occupation and that the working person is affected due to the listed disease. The third legal component is necessity of proving sufficient working period.

In case of Part A of Schedule III even a single day of exposure is enough, in case of Part B, six months working in that occupation is enough and in case of Part C the necessary periods are given in notifications.

The above periods when proven give the benefit to workmen.

If a working person contacts disease in lesser period or after retirement it is the duty of the working person to prove connection between the work and disease. In all other cases of Schedule III the connection is presumed.

SCHEDULE III

See section 3 of the Workmen's Compensation Act (1923) and Section 52 of the Employees State Insurance Act (1948)

S.No. (1)	Occupational Diseases (2)	Employment (3)
	PART A	
1.	Infectious and parasitic diseases contracted in an occupation where there is a particular risk of contamination.	a) All work involving exposure to health or laboratory work; b) All work involving exposure to veterinary work; c) Work related to handling of animals, animal carcasses, part of such carcasses or merchandise which may have been contaminated by animals or animal carcasses; d) Other work carrying a particular risk of contamination.
2.	Diseases caused by work in compressed air	All work involving exposure to the risk concerned.
3.	Diseases caused by lead or its toxic compounds.	All work involving exposure to the risk concerned
4.	Poisoning by nitrous fumes	All work involving exposure to the risk concerned
5.	Poisoning by organo - phosphorus compounds	All work involving exposure to the risk concerned

PART B

1.	Diseases caused by Phosphorus and its toxic compounds.	All work involving exposure to the risk concerned.
2.	Diseases caused by mercury or its toxic compounds.	All work involving exposure to the risk concerned.
3.	Diseases caused by benzene or its toxic compounds.	All work involving exposure to the risk concerned.
4.	Diseases caused by nitro or amido toxic derivatives of benzene or its homologues.	All work involving exposure to the risk concerned.
5.	Diseases caused by chromium or its toxic compounds.	All work involving exposure to the risk concerned.
6.	Diseases caused by arsenic or its toxic compounds.	All work involving exposure to the risk concerned.
7.	Diseases caused by radioactive substances and ionising radiations.	All work involving exposure to the risk concerned.
8.	Primary epitheliomatous cancer of the skin caused by tar, pitch, bitumen, mineral oil, anthracene, or the compounds, products or residues of these substances.	All work involving exposure to the risk concerned.
9.	Diseases caused by toxic halogen derivatives of	All work involving exposure to the risk concerned.

	hydrocarbons (of the aliphatic and aromatic series).	
10.	Diseases caused by carbon disulphide.	All work involving exposure to the risk concerned.
11.	Occupational cataract due to infrared radiation.	All work involving exposure to the risk concerned.
12.	Diseases caused by manganese and its toxic compounds	All work involving exposure to the risk concerned.
13.	Skin diseases caused by physical, chemical or biological agents not included in other items.	All work involving exposure to the risk concerned.
14.	Hearing impairment caused by noise	All work involving exposure to the risk concerned.
15.	Poisoning by dinitrophenol or a homologue or by substituted dinitrophenol or by the salts of such substances.	All work involving exposure to the risk concerned.
16.	Diseases caused by beryllium or its toxic compounds.	All work involving exposure to the risk concerned.
17.	Diseases caused by cadmium or its toxic compounds.	All work involving exposure to the risk concerned.
18.	Occupational asthma caused by recognised sensitising agents inherent	All work involving exposure to the risk concerned.

	to the work process.	
19.	Diseases caused by fluorine or its toxic compounds.	All work involving exposure to the risk concerned.
20.	Diseases caused by nitro-glycerine or other nitroacid esters.	All work involving exposure to the risk concerned.
21.	Diseases caused by alcohols and ketones.	All work involving exposure to the risk concerned
22.	Diseases caused by asphyxiants: carbon monoxide and its toxic derivatives, hydrogen sulphide.	All work involving exposure to the risk concerned.
23.	Lung cancer and mesotheliomas caused by asbestos.	All work involving exposure to the risk concerned.
24.	Primary neoplasm of the epithelial lining of the urinary bladder or the kidney or the ureter.	All work involving exposure to the risk concerned.

PART C

1.	Pneumoconioses caused by sclerogenic mineral dust (silicosis, anthraco silicosis, asbestosis) and silico-tuberculosis provided that silicosis is an essential factor in causing the resulting capacity of death.	All work involving exposure to the risk concerned.

2.	Bagassosis.	All work involving exposure to the risk concerned.
3.	Bronchopulmonary diseases caused by cotton, flax, hemp and sisal dust (Byssinosis)	All work involving exposure to the risk concerned.
4.	Extrinsic allergic alveolitis caused by the inhalation of organic dusts.	All work involving exposure to the risk concerned.
5.	Bronchopulmonary diseases caused by hard metals.	All work involving exposure to the risk concerned.

Infectious and Parasitic diseases contracted in an occupation where there is particular risk of contamination

Industrial occurrence	Disease
In industries associated with Abattoirs, agriculture, animal attendants, bone and bone meal processing, butchers, dairy, forestry, game wardens, hair, bristle processing, ivory, horn processing, laboratories, animal, meat packers, medical practitioners, medical workers, nurses, poultries, sewerage workers, stock farming, tanneries, veterinary work, wild life management, wood industry,	**Anthrax** **Brucellosis** **Infection of the mucous membrane of upper respiratory tract** **Erysipeloid** **Hepatitis B:** The percentages of affected doctors, medical workers is known to be 15 to 40 times more than the general population. **Hepatitis C** **Herpes simplex virus infections** **HIV infection** **Infective hepatitis** **Leishmaniasis (Kala Azar)** **Leptospirosis** **Mite infections** **Q fever** **Rabies** **Rat bite fever** **Ringworm** **Salmonellosis** **Toxoplasmosis** **Tuberculosis (TB)**

all occupations connected with animals (e.g. transport of animals).	

Diagnosis

1. Occupational history and clinical signs.
2. Standard tests for each disease.

Infectious bacterial skin diseases and surface infections

Industrial occurrence	Signs and Symptoms
In industries associated with Agriculture, animal breeders, animal handlers, bakers, bartenders, cannery workers, cooks, dairy workers, dental practitioners, dish washers, domestic workers, farmers, fishermen, food processors, grain handlers, harvesters, hide handlers, live stock handlers, live stock workers, longshoremen, medical practitioners, and silo workers, etc.	1. Staphylococcal and Streptococcal infections : most common are secondary infections from minor lacerations, abrasions, burns and puncture wounds. 2. Cutaneous tuberculosis - warty, granulomatous lesions usually over fingers, hands. 3. Tetanus 4. Paronychia ***Viral Infections*** 1. Orf - Macule --> papule --> Target lesion --> nodule with central umblication. 2. Milker's nodules due to hand milking (infected teats and udders of cattle); 1 cm , one or multiple nodules and regional lymphadenopathy present. 3. Cat scratch disease: Lichenoid - papular nodule at the site of recent cat scratch, regional lymphadenopathy three to four weeks later. 4. Viral warts ***Fungal Infection*** Dermatophytic infections:

	• Itchy lesions • erythematous plaques • scaling of palms and soles • onychomycosis 2 Candida infections: • Intertrigo of digital web spaces. • painful inflammation of periungual tissue, yellow- green nail discoloration. 3. Sporotrichosis: • Subcutanaous nodules that ulcerate and drain, lymph node enlargement. 4. Mycetoma: (Madura foot) 5. Blastomycosis 6. Coccidioidomycosis ***Parasitic Infestations*** 1. Cutaneous Leishmaniasis 2. Helminthic infections: Larva migrans (creeping eruptions) 3. Arthropods infestations: Mites, ticks, spider and scorpion bites. 4. Myiasis - Larva of flies invade human tissues; beetles, lice, bees, wasps, moths, ants, flies- all cause pruritic eruptions.

Diagnosis

1. Occupational history and clinical signs.
2. Standard tests according to particular disease.

Disease caused by work in compressed air; decompression sickness

Industrial Occurrence	Signs and Symptoms
In industries associated with Construction of foundations of bridges or piers; deep sea diving for construction and maintenance of barrages, cables, cooling water systems for power stations, effluent outfalls, harbours, immersed tube tunnels, ports, submarine pipelines; tunnelling under water; undersea houses and bases.	***Skin*** • Itching, usually in those parts which are exposed to compressed air and not immersed. • Blotchy red/ purple rash "cutis mazmorata" • "peau d'orange" oedema to parts. ***Musculo Skeletal*** • Pain in synovial joints mainly knee and shoulders • Pain in limbs, discrete/diffuse/mild/intense • These are occurrence of acute form of illness. • Bony necrosis usually in large joints, which may appear on X-ray even after 2 months of a single exposure. ***Constitutional symptoms*** • Malaise, anorexia, fatigue ***Pulmonary barotrauma*** • Chest pain, pneumothorax, gas in the mediastinum ***Cardiopulmonary decompression illness*** • Retrosternal pain, breathlessness, dry cough, shallow rapid respiration, circulatory failure.

Neurological

Any neurological symptoms / deficit within 36 hours of exposure should be considered due to decompression, unless proved otherwise. These are:

- Girdle pain
- Hemi/mono/para/quadri plegia
- Cerebral dysfunctions like visual disturbances, flashes of light/gaps in visual field etc.
- Sensory paradox (confusion of hot and cold)
- Loss of consciousness
- Occasional fits.

Ear and para nasal sinuses

- Acute pain in the ear and /or sinuses
- Oedema / congestion of ear drum
- Rupture of ear drum may occur.

Teed's classification of ear drum affection:

1. Congestion
2. Redness
3. Haemorrhage into tympanic membrane.
4. Haemorrhage into the middle ear may be with or without rapture of tymphanic membrane.

Chronic effect

Bony necrosis usually in large joints.

Diagnosis

1. Occupational history of exposure of work under pressure more than 1kg/square cm is sufficient when there is any of the symptoms/ complaints.
2. Radiographs of joints in chronic cases (osteonecrosis) once a year upto two years even after stopping work.

Diseases caused by lead and its toxic compounds

(Examples: inorganic compounds: almosite or lead silicate; anglesite or lead sulphide; galena or lead sulphide; lead peroxide; litharge or lead monoxide; red lead or triplumbic tetroxide; white lead or carbonate; etc. organic compounds:sugar of lead acetate; tetra-ethyl lead; etc.)

a) Inorganic compounds of Lead

Industrial occurrence	**Signs and Symptoms**
In industries associated with Alloys, ammunition, car repairing, ceramics, glazed potteries, inks, insecticides, lead lining, plumbing, printing press, radiator manufacture, rubber, (primary and secondary) smelting of lead, scrap metal work, storage batteries.	**Acute effects** 1. Lead colic 2. Lead encephalopathy • coma, delirium, convulsions, toxic psychosis, transient pariesis, aphasias, aphonia due to laryngeal nerve palsy, facial and occulo motor nerve palsy. • Acute forms : convulsions, delirium and coma **Chronic effects** ***CNS* :** • memory loss, headache, trembling, aphasia and hemianopia ***Visual changes* :** • Amaurosis • concentric diminution of visual fields • papilloedema • optic atrophy

	Neurological changes **:** • wrist drop- • foot drop in severe cases ***Hypertension*** ***Chronic nephritis*** ***Impaired fertility*** ***Stillbirths*** ***Anaemia of lead poisoning*** ***Lead colic***

Diagnosis

1. Occupational history and clinical signs.
2. Hypochromic microcytic anaemia and stippled basophillia.
3. Pb in urine.
4. Pb in plasma.
5. Pb in urine after EDTA-Ca infusion.
6. Urinary aminoleunic acid.
7. Urinary coproporphinogen.
8. Lumber Puncture in acute cases, shows increased pressure and lymphocytes on CSFexamination.

b) Tetra-ethyl lead

Industrial occurrence	Signs and Symptoms
In industries associated with Gasoline fuel for internal combustion engines, (as `anti-knock' agent), refineries.	Mild manifestations - insomnia, lassitude, nervous excitation, lurid dreams in association with tremors and spasmodic muscular contractions. More severe responses - Complete disorientation with hallucinations, facial contortions, episodes of hallucinations may be converted into maniacal or violent convulsive seizures which may terminate in coma or death.

Diagnosis

1. Occupational history and clinical signs.
2. Increased levels of lead in urine.
3. Colic, palsy and stippling are absent.

Diseases caused by nitrous fumes

(Examples: di-nitrogen trioxide; laughing gas, nitrogen dioxide, nitrogen monoxide, nitrous oxide; di-nitrogen pentoxide; etc.)

Industrial occurence

In industries associated with

Bleaching of rayon (as stabiliser); nitric acid manufacture, welding (and as by-product and intermediate product in many industries)

Signs and symptoms

Acute effects

1. Nitric oxide and nitrogen dioxide are powerful lung irritants.
2. High concentrations may cause sudden death.
3. Death may be caused due to delayed oedema of lungs
4. Initial exposure may be no more than mild irritation of eyes and the respiratory tract.
5. After initial exposure the symptoms gradually subside over 2-3 weeks . Then they may enter the 2nd stage with increasing severity, fever with chills that may precede a relapse, with increasing dyspnoea, cyanosis and recurring pulmonary oedema.

Chronic effects

Drowsiness, dizziness and vomiting associated with the presence of methaemoglobin in blood

Lung function may be affected

Diagnosis

1. Occupational history and clinical signs.
2. Chest X-rays: multiple discrete nodules
3. Blood test for methaemoglobin.

Diseases caused by organo-phosphorus compounds

(Examples: Alkron; Chlorpyriphos; DDVP; Diazion; Diomethoate; DNTP;DPP; Ethion; Gliphosate; Gusathion; Malathion; Monotriphos; Phos-Kill; Phosphomidon; Sumithion; Thiphos or Parathion; Trichlophon; and similar pesticides)

Industrial occurence	**Signs and symptoms**
In industries associated with Agriculture,use of above pesticides; organo-phosphorus pesticides production; pest control.	**Acute effects** ***CNS*:** Salivation * Incontinence* Sweating Convulsion Headache Nausea Dizziness Restlessness, anxiety Toxic psychosis, delirium Unconsciousness ***Musculo skeletal system*:** Muscle twitching * Faciculation* Sweating Weakness Incoordination Tremor Paralysis

GIT System
Diarrhoea
Vomiting
Abdominal cramps
Vision
Miosis*
Blurred vision
Watering of eyes
Respiratory tract
Rhinorrhea*
Pulmonary oedema*
Bronchorrhea*
Wheezing, chest, tightness*
Respiratory paralysis
CVS
Predominant bardycardia (parasympathetic stimulation)
Sinus arrest
Early Tachycardia (Sympathetic ganglia stimulation)
Early hypertension (Sympathetic ganglia stimulation)

* Key aspects of syndrome.

Chronic effects

1. Delayed neuropathy:
   Muscle weakness, fatigue, flaccid paralysis
2. Behavioural effects
3. Cognitive effects ,vigilance, information processing and psychomotor skills and memory, speech; both performance and perception are affected, psychic state; increased tendencies to depression, anxiety and irritability.

EEG: Faster frequencies and higher voltages

Diagnosis

1. Occupational history and clinical signs.
2. Erythrocyte and/or pseudo cholinesterase level may be decreased (rate of decrease is more indicative)
3. ECG changes.

b) Triortho cresyl phosphate

Industrial occurrence	**Signs And Symptoms**
In industries associated with Used as plasticizer in vinyl plastics, flame retardent , solvent for nitrocellulose, additive to extreme pressure lubricants, non-flammable fluid in hydraulic system etc.	**Acute effects** Nausea, vomiting, abdominal pain and diarrhoea Latent period 3-28 days Delayed neurotoxicity Cramp like pains in calves numbness and tingling in feet ---------> weakness lower limbs ---------->> foot -drop ---------->> wrist -drop. initially lower - motor neuron type lesion later spasticity and pyramidal signs, --> muscle wasting. **Chronic effects** Weakness of muscles persist

Diagnosis

1. Occupational history and clinical signs.
2. Erythrocyte and/or pseudo cholinesterase level may be decreased (rate of decrease is more indicative)
3. ECG changes.

Diseases caused by phosphorus and its toxic compounds

(Examples: Calcium superphosphate; Orthophosphoric acid or phosphoric acid; Phosphine, hydrogen phosphide or phosphorated hydrogen; Phosphorus pentachloride; Superphosphates and Phosphates; Tetra-phosphorus trisulphide; Zinc phosphide; etc)

a) Diseases caused by Phosphorous

Industrial Occurrence	**Signs and Symptoms**
In industries associated with Chemicals; detergents; explosives; fertilisers; fireworks, ignition compounds; incendiaries; insecticides; iridescent metallic deposits; phosphorus bronze; rodenticides; rust-proofing of metals; safety matches.	**Acute effects** Clinical manifestations can be seen in four stages in cases of accidental ingestion. Stage I - Latent period ; few minutes to 6 hours Stage II - Relates to gastro - intestinal symptoms. • It comprises of garlic like taste, burning pain in throat and stomach, and intense thirst. This is followed by marked dehydration, nausea and vomiting. • Some times diarrhoea ensues. In more severe cases peripheral circulatory failure sets in. Stage III - It is marked by a second latent period. The symptoms abate for 2 to 4 days and there is a semblance of recovery. Stage IV - This resembles acute viral hepatitis. In this stage Hepatic failure

	ensues. Local irritant effects on skin and mucous membranes and respiratory tract leading to bronchitis and acute bronchitis or pulmonary oedema. Skin burns are possible. **Chronic effects** Symptoms are pain in abdomen, salivation, nausea, vomiting, diarrhoea, hepatitis, irritation of respiratory tract. Pain in joints, toothache, inflammation of jaw bone, necrosis and failure of dental sockets to heal when removed are most characteristic. Mucous membrane of the mouth gets inflammed and shows small reddish areas. **Phosphates and other compounds** Metaphosphates may be highly toxic causing discharge of blood and damage to liver and kidney. Workers working with phosphates and superphosphates may have problems of flurosis (see B-19) and hazards due to radioactivity (see B9) due to contamination of phosphates.

Diagnosis

1. Occupational history and clinical signs.
2. Regular X-ray of teeth, liver function test for cellular damage.

b) Diseases caused by Phosphine

Industrial Occurence	Signs and Symptoms
In industries associated with Fertiliser use and production; fumigation of grain (phosphine is produced in action of aluminium phosphide with water) ; mining and processing of phosphates; phosphoric acid production.	**Acute effects** Depression of central nervous system, restlessness, tremors, fatigue, headache, severe gastric pains, oedema of lungs. **Chronic effects** Symptoms appear after continued exposure even to low concentrations - anaemia, bronchitis, disturbance in vision and speech and also in muscle movements.

Diagnosis

1. Occupational history and clinical signs.

Diseases caused by mercury and its toxic compounds

(Examples: inorganic compounds: mercuric azide; mercuric chloride; mercuric oxide; mercuric sulphide; mercurous azide, mercurum; mercury cyanate; mercury fulminates; organic compounds: MEMC or methoxy ethyl mercuric chloride; methyl mercuric chloride; PMA or phenyl mercuric acetate; etc.)

a) Mercury and inorganic compounds of mercury

Industrial Occurrence	**Signs and Symptoms**
In industries associated with Acetaldehyde and acetylene; acetic acid; agricultural and industrial poisons; amalgams manufacture; antifouling paint; artificial silk; barometers; chloralkari process for chlorine production detonators; dental amalgams; electrical apparatus; incandescent bulbs; mercury vapour tubes; rectifier batteries; silver	**Acute effects** Most common where metallic mercury is handled in commercial quantities in an enclosed space i.e. cleaning out vessels and tanks as an electrolytic cell. • There is metallic taste in the mouth, constriction in throat, hoarsensess of voice and difficulty in breathing. • The features of interstitial pneumonitis may occur. • The mouth and tongue become corroded, swollen and coated with greyish white coating. • Hot burning pain is felt in the mouth and stomach. • There may be nausea and vomiting followed by blood strained diarrhoea. • Psychotic reactions - delirium, hallucinations and suicidal tendency

ores; textiles; thermometers; treatment of gold; vaccum pumps and x-ray tubes; laboratory equipments; switch gears.	• Nephrotoxicity : Protinuria followed by Nephrotic syndrome **Chronic effects** In elemental and inorganic form - Nervous & G.I. System are affected more. In Organic form - Erethism is more common. **G.I. System** Gingivitis, excessive salivation, digestive disturbances, colicky pain, vomiting and diarrhoea may be the first symptoms. There may be glossitis and pharyngitis. Loosening of teeth may occur. **Nervous System** • Tremor may be the first sign or presentation as change in hand writing. • Tremor starts with hands and fingers then spread to eye lids, lips and tongue. Intentional tremor is first seen . • Subsequently it is present at rest too, but disappears with sleeping. • Loss of memory, vivid dreams and night mares. • Spastic gait.

Diagnosis

1. Occupational history and clinical signs.
2. Urine examination for mercury.
3. Blood examination for mercury.

b)Methyl Mercury compounds and other organic compounds of mercury

Industrial Occurrence	**Signs and Symptoms**
In industries associated with Antiseptics, germicides, diuritics, contraceptives, and pesticides; preservative in paints, waxes, pastes, antifouling paints, and used in organic syntheses.	• Sensory, visual, auditory and cerebella affections. • Earliest: paraesthesiae, malaise, blurred vision. • Later : constriction of visual fields, deafness dysarthria, ataxia leading to neuromuscular weakness. • Foetus : microcephaly, hyperreflexia, mental impairment, blindness and deafness. • Methyl mercury affects cerebellum causing cerebella ataxia. • Along with this feature of erethism, a conglomeration of features of psychosis, may be present. • Erethism is characterised by extreme timidity, irritability, sudden out burst of temper, loss of memory, dysarthla etc. i.e. change of personality. There may be features of kidney diseases like Nephorotic Syndrome. ***Skin :*** Contact dermatitis (Pink disease) and sarcoid like granuluma

Diagnosis

1. Occupational history and clinical signs.

Diseases caused by benzene and its toxic homologues

(Examples: Toluene; Xylene; etc.)

a) Benzene

Industrial Occurrence	Signs and Symptoms
In industries associated with Chemical syntheses (widely used as fuel, chemical reagent, solvent, additive in motor fuel, and raw material, especially for organic chemicals) and tanning etc. *Manufacture of* : artificial manure; cyclohexane; detergents; glue; paint removers; pesticides; phenol. shoes and styrene.	**Acute effects** • Acute narcotic action producing unconsciousness convulsions and paralysis leading to coma and death. • Local irritant effect on skin and mucous membrane and G.I. tract. **Chronic effects** (Haematological changes) • Anaemia, macrocytosis, thrombocytopenia and leukopenia. • Retic count, increased. • Eosinophilia. • Immature cells in circulation. • Evidence of haemolysis. • Occasional leukocytosis. • Bone Marrow may be acellular or hyperplastic. • Leukaemias : initially leukopenia. acute myeloid leukaemia, acute monocytic leukaemia, chronic myeloid leukaemia.

If exposure is not terminated true aplastic anaemia may develop with partial or total destruction of all elements of bone marrow.

Estimated percentage of worker population that might develop toxicity after Benzene exposure			
Duration	Exposure	Bone marrow	Aplastic Anaemia
1 year	100 ppm	90	10
	50 ppm	50	5
	10 ppm	1	0
	1 ppm	0	0
10 years	100 ppm	99	50
	60 ppm	75	10
	10 ppm	5	0
	1 ppm	<1	0

Other long term effects of Benzene
1. Immunotoxicity
2. Chromosomal aberrations

Diagnosis
1. Occupational history and clinical signs.
2. Complete blood count.
3. Peripheral smear of blood.
4. Bone marrow examination.
5. Urinary phenol.

b) Toluene

Industrial occurrence	**Signs and Symptoms**
In industries associated with Benzene; as solvent for paints and coatings; as a component of gasoline.	**Acute effects** Same as benzene **Chronic effects** 1. Skin : dryness, contact dermatitis. 2. CNS : headache, lassitude, loss of appetite, impairment of coordination, loss of memory, headache, palpitation and severe weakness. 3. Liver - hepatomegaly, altered liver function. 4. Haematological - Macrocytosis. 5. Other : Menstrual disturbances. Chromosomal aberrations.

Diagnosis

1. Occupational history and clinical signs.

Diseases caused by nitro and amido toxic derivatives of benzene or its homologues

(Examples: Nitro-aniline; Nitrobenzene; Nitrotoulene; o-Amino phenol; o-Choloro aniline; o-Nitro-chlorobenzene; o-Toluedine anisidine; p-Amino phenol; Toluedine; etc.)

Industrial Occurrence	Signs and Symptoms
In industries associated with Antioxidants; dyes, elastomers, explosives, fuel additives, pharmaceuticals, pigments, plastics, resins, rubber accelerators, solvents and textiles. Large quantities of Nitrobenzens are converted to aniline by the reduction of the Nitro group, the aniline in turn serving as the starting point for a great number of industrial chemical reactions and synthesis	**Acute effects** • Onset is insidious and cyanosis appears when methhaemoglobin level in blood reaches 15% or more. • In later stages hypotension, headache, nausea and numbness of the limbs occur. In some cases excitement and tremors followed by severe depression, unconsciousness and coma may be seen. **Chronic effects** Anaemia, perhaps headache, fatigue, nausea, chest pain, numbness, nervousness, difficulty in breathing. • Repeated exposure affects liver causing yellow atrophy and varying degree of anaemia. • Peripheral neuropathy • Allergic dermatitis may also be seen. • Skin irritation in case of nitro-chloro-benzenes; dermatitis in case of slight

particularly in dye-stuff industries.	contact with di-nitro-chloro-benzene. • 2-4-di-nitro-toluene affects liver • Aniline and some nitro compounds are suspected carcinogens. • Aniline causes liver toxicity and cancer of urinary bladder.

Diagnosis

1. Occupational history and clinical signs.
2. Methemoglobin level.
3. Liver function test.

Diseases caused by chromium and its toxic compounds

(Examples: Crocolite chromate; Chromates; Chrome yellow; Chromic acid/Chromium trioxide; Chromium carbide; Chromium hydride; Chromium sulphate; Potassium dichromate/bichromate, Red potassium chromate; Sodium dichromate, acid chromate; etc.)

Industrial Occurrence	**Signs and Symptoms**
In industries associated with Chromium plating, chromium salts, leather tanning, metallurgy, photomechanical processing and refractory bricks. *Chromium in valency six (in chromates chromium has valency six) is more hazardous than valency three. In valency three compounds it is easily absorbed in the intestine.*	**Acute effects** • Toxic effects may appear in few minutes. • The symptoms include bitter metallic taste, intense pain in abdomen, vomiting and diarrhoea. • Stools may contain blood. • There is oliguria and anuria. • These are followed by muscular cramps, mental confusion, cyanosis, collapse and unconsciousness. • Death may occur due to uraemia. **Chronic effects** *Primary dermal irritation* Hyperaemia; papiulo, vesicular rash, ulcers due to contact with all chromium products. Punched out ulcers: nail root areas, creases of knuckles, finger webs, backs of hands and forearms are the more common sites.

	Allergic contact dermatosis. Dermatitis and eczema. caused by cell mediated immunity. Patch tests : Positive. Also positive photosensitivity tests. *Perforation of nasal septum.* *Effects on lung : generalised obstructive lung disease,* FVC and FEV_1 reduced and occupational asthma. *Renal* : Nephritis Tubular necrosis Urine albumin, hyaline/ granular casts and presence of RBCs in urine. *G.I. : hyperchlorhydria and peptic ulcer* *Cancer* : lung sinonasal buccal cancer. pharyngeal oesophageal cancer.

Diagnosis

1. Occupational history and clinical signs.
2. Blood, urine analysis (post shifts).
3. Patch tests.

Diseases caused by arsenic and its toxic compounds

(Examples: Aminopheyl arsenic acid; Antoxylic acid or arsenalic acid; Arsenic sulphide; Arsenic trichloride; Arsine; Calcium arsenite; Lead arsenite; Schill's mineral or cupric arsenite; White arsenic or arsenic trioxide; etc.)

Industrial Occurrence	**Signs and Symptoms**
In industries associated with Drugs manufacture (indigenous as well as other systems of medicine); fungicides; glass; insecticides; metallurgy, in hardening of copper, lead and other alloys; pigment production; preservation of hides, skin, fur, wood, etc.; rodent poison, smelting of copper ores (as by-product).	**Acute effects** Mostly occur after accidental ingestion of arsenic. Characterised by profound gastro intestinal disturbances, resulting in severe diarrhoea and vomiting which may result in shock and subsequent oliguria and albuminuria. There may be facial oedema, muscle cramps and cardiac abnormality. Acute intoxication with arsenic compounds is accompanied by anaemia and leucopaenia. Reversible enlargement of liver is also found. In individual surviving acute poisoning peripheral nervous disturbances frequently develop a few weeks after ingestion. After a spill of arsenic trichloride, fatality has been reported. Some organic arsenicals, such as arsenilates, have a selective effect on the optic nerves and can cause blindness. Increased mortality due to lung cancer has been reported.

Chronic effects

Chronic arsenic poisoning may occur in workers exposed for a long time to excessive concentration of airborne arsenic compounds.

Local effects on the mucous membranes of the respiratory tract and skin are prominent features. Involvement of the nervous and circulatory system and the liver may also occur as well as cancer of the respiratory tract may occur.

Skin

1. It forms hyperpigmentation which is called 'Rain drop pigmentation'. mostly over the face and neck. To start with fine discrete mottled brown pigmentation is seen. In long standing cases skin of the back may be affected and may become bronze coloured.
2. Follicular eczematous dermatitis.
3. Symmetric verrucous hyperkeratosis of palms and soles.
4. Melanosis with depigmented spots.
5. Hyper pigmentation of sole, feet and palms.
6. Ulceration of skin.
7. Basal cell cancer or Squamous cell cancer.

Respiratory System

Irritation of mucous membranes lead to pharyngitis, laryngitis, glossitis. Perforation of nasal septum may occur within a very short time as a result of deposition of arsenic. It is usually painless.

	Liver Enlargement of liver with features of cirrhosis of liver, non cirrhotic portal fibrosis with portal hypertension. ***Blood*** It inhibits haem synthesis causing megaloblastic anaemia ***Nervous System*** Peripheral neuropathy characterised by paraesthesis in glove and stocking distribution and loss of vibration sense. There may be pain, burning sensation, pain over the calf muscle causing difficulty in walking. **Following cancers are reported** • Lung • Skin • Liver - Malignant haemangio endothelioma • Leukaemia • Oesophageal • Urinary bladder • Lymphatic system.

Diagnosis

1. Occupational history and clinical signs.
2. Estimation of arsenic in urine.
3. Liver function test.
4. Estimation of arsenic in hair and nails may be useful (levels need to be standardised), but its value in industrial exposure is questioned.

Diseases caused by radioactive substances and ionising radiations (X-rays, etc.)

Industrial Occurence	Signs and Symptoms
In industries associated with Aerosol fire detectors; gas chromatography, industrial radiography, nuclear reactors, radioactive tracers, radium dial painting; uranium mining; use and analysis, or manufacture of radioactive materials and x-ray clinics.	• Exposure of the entire body or large portion of the body to doses of radiation in excess of 1 Gray (Gy) results in nausea, vomiting, perhaps diarrhoea, within hours of exposure. This is the first phase. • Symptoms relating to stomach and intestines improve in a day after which the second phase starts - a period of relative well being that may last upto one week. • The third phase is characterised by recurrence of intestinal symptoms, ulcers in mouth and throat, possible loss of hair, and possibly gross bleeding in gastro-intestinal tract. • Pancytopenia and immunosupression, in case of localised exposure abnormal formation of fibrous tissues may take place in spaces within organs or tissues. • Exposure to high doses and also to repeated small doses may lead to cataract after a latent peniod of usually six months to two years but in rare cases it may extend even upto twelve years. • Exposure to cumulative doses in excess of 3000 to 6000 rads gives rise to radioactive hepatitis, 2 to 6 weeks later

	sclerosis may develop. • If embryo is exposed - morphological abnormalities in development of nervous system or even death of embryo of fetus is possible, depending upon the dose and duration of exposure. • Local tissue damage : Skin; radiation dermatitis , eczema, epilation and atrophy. • Gonads : Genetic mutations after more than 10 rads exposure • Cancer of the following organs: Thyroid Bone Skin Lung Liver and other organs.

Diagnosis

1. Occupational history and clinical signs.
2. Blood count.

Dr.Meneges (name changed on purpose) aged 24 years and doing his residency in orthopaedics in a major hospital complained of severe fatigue weakness and pallor.
He had history of helping patients to take X-rays for 6 months.

On investigation :

Hb	:	*5.4*
Haemato count	:	*18*
Total Count	:	*2,800*
Platelets	:	*15,000*
Reticulocyte count	:	*12*
ESR	:	*84*

On bone marrow examination

Myeloid series : depleted, no blasts

Erythroid series : Depleted *cont.*

Megakayocytes : Inadequate
Diagnosis : Severe Hypocellular marrow

He was treated and was asked to avoid radiation exposure. After 10 months of treatment his Hb was 9 gm% and platelet count 25,000 and WBC count is adequate. Dr. Meneges left the job and after a court battle he has taken up residency in general surgery.

Final diagnosis : Radiation induced occupational aplastic Anaemia. He is on treatment with cyclosporine (cost Rs.400/- per day) and expected to be on treatment for 5 years.

Assessment of disability : (based on blood tests done 2 times in a month in spite of treatment.)

1. *Due to anaemia : 25%*
2. *Due to WBC depletion : 50%*
3. *Due to platelet depletion : 100 %*

Disability : 100% of whole man

Kindly refer to `Impairments, Disabilities and their Assessment', Published by PRIA, New Delhi.

Primary epitheliomatous cancer of the skin caused by tar, pitch, bitumen, mineral oil, anthracene, or their compounds, products or residues

Industrial Occurence	Signs and Symptoms
In industries associated with Anthracene (used in dyes, and radiation detection); asphalt; coal, gas works, mineral oils (i.e., oils found in rock strata, e.g., petroleum).	The skin lesions are marked by keratosis, papillomata and epetheliomata. There may be increase or decrease of pigmentation in the epidermis. Fissures may be found with secondary infection of the skin. Tumour appears as a small but firm aggregation of cells having diffused redness.

Diagnosis

1. Occupational history and clinical signs.
2. Skin lesion.
3. Biopsy report.

Diseases caused by halogen derivatives of hydrocarbons

(Examples: Bromo benzene; Bromo methane; Carbon tetrachloride; Chloro methane or methyl chloride; Chloroform; Chloroprene or Chlor-butadiene, 2-chloro-1-3-butadiene or fluro-trichloro methane; Freons; Vinyl chloride; etc.)

a) Diseases caused by Vinyl Chloride

Industrial occurrence	Signs and Symptoms
In industries associated with Production of Vinyl chloride resins (PVC), copolymers, methyl chloroform; component of propellent mixtures.	**Acute effects** • Dizziness. • Somnolence. • Euphoria. • Narcosis. • Loss of conciousness. **Chronic effects** • Hepatomegaly. • Hepatic fibrosis. • Angiosarcoma of liver and other carcinomas. • Portal hypertension. • Haemangiosarcomas. • Bile duct cancer. • Malignant tumours of brain and CNS. • Raynaud's phenomenon. • Oligospermia may result in fetal death.

b) Fully halogenated chlorofluorocarbons (CFS)

Industrial occurrence	Signs and Symptoms
In industries associated with Chemicals (as solvents, refrigeneants, anaesthetics, fumigants, etc.); Gauge fluids and Plastic intermediates.	**Acute effects** • Tingling sensation, humming in the ears, apprehension, EEG changes, slurred speech, significant degree of cardiac arrythrrias Fatalities are possible **Chronic effects** • Pain in legs. • Paraesthesia, weakness in legs • Decreased motor conduction velocity. • Palpitations. • Cardiac arrythrrias.

c) Carbontetrachloride (CCl_4)

Industrial Occurrence	Signs and Symptoms
In industries associated with Solvent for oils, fats, lacquers, varnishes, rubber, waxes and resins; used for degreasing and cleansing and as fire extinguisher.	**Acute effects** Acute nausea vomiting, drowsiness, dizziness and oliguric renal failure. **Chronic effects** Dysentry, dermatitis, liver damage (centri-lobular necrosis with or without fatty degeneration)

Diagnosis

1. Occupational history and clinical signs..
2. X-ray of fingertips in case of vinylchloride.
3. Liver function test.
4. Renal function test.

Uses and toxic properties of some industrially important aliphatic and aromatic halogenated hydrocarbons

Substance	Main uses	Degree of toxicity		
		Target organ or tissue	Acute	Chronic
Methyl chloride (CH_3Cl)	Chmical syntheses (methylation), refrigerant special extraction agent	CNS liver	High mild	High
Methylene chloride (CH_2Cl_2)	Solvent (oils, fats, waxes, cellulose acetate) degreaser, paint remover	CNS	mild	mild
Chloroform ($CHCl_3$)	Solvent (lacquers), extraction agent	CNS liver	High moderate	mild mild
Carbon Tetrachloride (CCl_4)	Chemical syntheses, fire extinguishers (for use as a solvent should be replaced by less toxic substance)	CNS liver Kidneys	moderate high high	mild high moderate
Ethyl chloride (CH_3CH_2Cl)	Chemical syntheses, local anasthetic (freezing)	CNS	moderate	Not known
1,2-dichloroethane ($ClCH_2CH_2Cl$)	Chemical syntheses, solvent (resins, rubber, bitumen, paints), degreaser, extraction agent for oils etc.	CNS liver kidney	moderate	mild mild
1,1,1-trichloroethane	Degresing agent in metal cleaning and drycleaning; good substitute for carbon tetrachloride	CNS	high	moderate
1,1,2-trichloroethane ($CH_2ClCHCL_2$)	Solvent (should be replaced by less toxic substances)	CNS liver kidneys	moderate high high	mild high moderate
tetrachloroethane ($CHCl_2CHCl_2$)	Solvent (should be replaced by less toxic substances)	CNS Liver kidneys	moderate high high	mild high moderate
trichloroethylene ($ClCH=CCl_2$)	Degreaser, drycleaning and extraction agent; chemical syntheses	CNS	high	high

tetrachloroethylene ($Cl_2C=CCl_2$)	Degreaser, drycleaning and extraction agent, chemical syntheses	CNS	high	mild
vinyl chloride ($CH_2=CHCl$)	Intermediate in the manufacture of poly vinylchloride	CNS liver bones	mild	 high high
methyl bromide (CH_3Br)	Insect fumigant (grain), chemical syntheses	CNS	high	high
ethyl bromide (CH_3CH_2Br)	Chemical syntheses, special extraction agent	CNS	moderate	Not known
1,2-dibromoethane ($BrCH_2CH_2Br$)	Insect fumigant (soil), fire extinguisher, solvent (celluloid, fats, oils, waxes)	CNS liver kidneys	mild moderate mild	mild mild mild
chlorobenzene ($C_6H_5Cl_2$)	Chemical syntheses, solvent	CNS liver mucous membranes	moderate mild mild	Not known
dichlorobenzene ($C_6H_4Cl_2$)	Chemical syntheses, insecticide	CNS liver mucous membranes	high moderate moderate	moderate moderate

Diseases caused by carbon disulphide

Industrial Occurence

In industries associated with Industrial solvents (widely used for alkalis, cellulose, fats, oils, resins and waxes).

Manufacture of Artificial silk, by viscose process; oil (by extraction); optical glass; pesticides (is itself also used as a pesticide).

Signs and Symptoms

Acute effects

- Fatalities are possible.
- Extreme irritability.
- Uncontrolled anger.
- Rapid mood changes.
- Maniac delirium, hallucinations.
- Paranoia.
- Suicidal tendency.
- Skin and muscle membrane irritation.

Chronic Effects

- Skin : blisters due to irritant dermatitis.
- Ophthalmological effects:
  Changes in :
  the motility of eyelids,
  the sensitivity of cornea and conjunctiva,
  the motility of eye ball,
  convergence and accommodation.
  optic nerve atrophy
  retrobulbar neuritis,
  sclerosis of fundic blood vessels,
  focal haemorrhage are possible.

Otological effects :
hearing impairment
reduced excitability of vestibular apparatus manifested as vertigo.

- Respiratory irritation.
- Gastro intestinal effects :
  gastritis leading to atrophic gastritis,
  duodenal ulceration,
  liver and bile duct dysfunction,

- Liver effects : Fatty degeneration of liver.
- Renal effects :
  Glomerulosclerosis, nephrotoxicity,
  arteriosclerotic injury to kidney
- Haematological effects :
  anaemia,
  slight reticulocytosis,
  eosinophilia,
  hypercoagulability of blood,
- Endocrine system :
  hypogonadism (decreased exerction of 17 ketosteroid and 17 hydroxy-ketosteroid in urine)
  hypothyroidism,
  reduction in adrenal activity.
- CNS : headache,
  Memory impairment,
  Rapid mood changes,
  Insomnia,
  Paraesthesia,
- General weakness and muscle pain and fatigue.
- EEG : episodic theta activity or diffuse abnormality,
- Peripheral nervous system
  Paraesthesia,

	Dysaesthesia, Fatiguability and diffuse pains in limbs, Symmetrical hyporeflexia and symmetrical polyneuropathy, Motor paresis, EMG - Reduces number of motor unit, recruitment • CVS effects : Vasculopathy, Accelerated atherosclerosis, Myocardial infarct, Hypertension. • Other effects : Hypercoagulability of blood, Teratogenic effects.

Diagnosis

1. Occupational history and clinical signs.
2. 2 or 3 medical examinations annually

Surveillance tests

1. Clinical neurological examination.
2. EMG studies.
3. Psychological and behavioural testing.
4. Serum T3,T4, TSH levels.
5. ECG.
6. Fundoscopy.
7. Lipid profile.
8. EEG.
9. Iodine - azide test in urine.

Occupational cataract caused by infrared radiation

Industrial Occurence	Signs and Symptoms
In industries associated with Arc processes; hot furnaces; lasers; molten glass; molten metals; presence of infrared radiation.	Lens of the eye or the capsule of the lens or both become opaque. Where eyes are exposed to processes hot enough to be luminous, the lens of the eye is more affected and the term 'glass workers cataract' is often used for this type of cataract.

Diagnosis

1. Occupational history and clinical signs.
2. Eye examination at regular intervals.

(a) Occular diseases caused by nonionising radiations not covered by schedule III

Sources	Effects	Occupation
Sunlight	Cataract Sunburn Solar retinitis	Outdoor workers (farmers, other farm workers etc.), Construction.
Arc lamps cameras (Xe, XeHg)	Photokeratitis retinal injury	Printing plants Camera operators, Optical labs.
Carbon arcs	Photokeratitis	Certain lab workers, Search light operators.
Lasers	Retinal injury	Construction
Argon lasers	Retinal injury	Lab workers, medical personnel.

Diseases caused by manganese and its toxic compounds

(Examples: Manganese salts; MMT or methyl cyclopentadynil manganese- tricarbonyl; Permanganate salts; Potassium permanganate; Pyrolusite or manganese dioxide; etc.)

Industrial Occurence	Signs and Symptoms
In industries associated with "Anti-knock' agent in petrol; ceramics; driers of linseed oil; dyeing and bleaching of textiles; electrode coating of welding rods; glass, inks; mining of manganese; paints and pesticides; tanning of leather; (production of) aluminium alloys; copper alloys; dry cell batteries; fertilisers; manganese compounds; potassium permanganate and steel and alloy	Damage is reversible if patient is removed in early stage but become more sensitive to further exposure. 3 stages are seen: (a) A prodermal stage : Anorexia, asthenia, somnolence, insomnia, decreased libido, headache. (b) Early clinical stage : Onset of extrapyramidal symptoms Speech disturbances leading to mutism Increased tone of facial muscles with masked facies. Decreased ability to perform skilled movements Exaggerated tendon reflexes (c) Established chronic poisoining : Marked rigidity more in lower limbs and face Asthenia, muscle pain, paraesthesiae, intentional tremor, no resting tremor, dystonia (instability of complimentary muscle groups) Apathy, unmotivated laughter, a tendency to weep, irritability, increased salivation. Gait distubance - first apparent as 'difficulty

steels.	in stepping backwards. Reduced immunity to respiratory infections resulting in • Repeated Pharyngitis • Lobar pneumonia • Chronic bronchitis.

Diagnosis

1. Occupational history and clinical signs.
2. Urine, blood, faeces analysis.
3. Information should be collected from friends, colleagues and relatives about behavioural changes.

Skin diseases caused by physical, chemical or biological agents not included in other items

Skin diseases are reported in almost all occupations

a). Skin diseases caused by physical factors

Industrial Occurence	Signs and Symptoms
In industries associated with Cold; contact with liquid tar; dyes (those activated due to certain wavelengths of light); electric furmances; electricity; heat; high energy sources such as x-rays; humidity; metal burning; molten metal pouring; pipeline work; plasma torch burning; sunlight; ultraviolet rays (artificial) and welding.	(1) Mechanical Trauma :- It accounts for 6% of cases.

Occupations	Skin manifestations
Tannery stakers	Knuckle calluses
Diamond stakers	Knuckle pads
Violinsts	Folliculitis, lichenification
Coal miners	Tattooing
Bakers	Bakers' sinus(inflammed interdigital cleft)
Blacksmiths fingers	Bullae on palms and
Butchers	Palmer hyperkeratoses

(2) Heat: - Occurrence commonly seen in Bakers, boilers, heaters, asphalt workers, foundry workers, firemen, miners, etc.

Diseases: Miliaria, intertrigo, erythema abigne, urticaria and cancer.

(3) Cold :
(a) *Diseases due to abnormal cold*:- frostbite, immersion or trench foot.
(b) *Diseases due to abnormal reaction to cold*: chilblain, acrocyanosis, erythrocyanosis, livedo reticularis, cryoglobulinaemia, cold urticaria, Raynaud's phenomanon.

(4) Humidity: Low: Occurrence commonly seen in workers working in soft contact lens factories, silicon chip manufacture and cabin crews of long distance airplanes.
Diseases: Pruritus, burning, erythema and scaling.

(5) Vibrations: Occurrence commonly seen with chain sawers, elecrtical grinders, miners, pneumatic tool operators.
Diseases: Reynaud's phenomenon.

(6) Radiation: (a) Acute Effects: Sun burns, burns.
(b) Chronic Effects: Polymorphos light eruption
(c) Chronic Cumulative Effects: Premature ageing, keratoses, cutaneus cancers.
(d) Photosensitive Reaction: Phototoxic or photoallergic dermatitis.
(e) Lasers; Cutting and drilling metalglass or diamond, medical fields (opthamology and

	dermatology) - Cataracts.
	Contact with electric short circuits or defective electrical apparatus causes burns and destruction of deeper tissues.

b) Skin diseases caused by chemicals

Chemicals causing skin diseases are classed as **primary irritants** and **sensitizers.** 'Primary irritant' is a chemical that injures skin with sufficient exposure. Some - such as concentrated acids and alkalies, metallic salts, some solvents, and some gases - cause injury very rapidly; others may require several days of repeated contact to produce observable effects.

'Sensitizer' is a chemical that causes skin of a susceptible individual to react. Persons who are allergic to particular sensitizer are affected by it. Even very small amount of chemical, otherwise rated as harmless in that quantity for the general population, may sensitize susceptible individuals. Below is a list of the most common irritants and sensitizers and some related information (Please see the appendix for detailed list) .

Primary Irritants

Abrasives, acids, alkalies, cement, chlorinated diphenyls, detergents, disinfectants, dyes, fibrous glass, gum, hardneres, inks, lime, nitro-paints, organic solvents, peroxides, pesticides, soaps, synthetic coolants, turpentine and weed-killers, etc.

Sensitizers
Azo dyes, chromium, epoxy resins, fibrous glass, formaldehyde, fungicides, mercury and cobalt salts, nickel and, turpentine, etc.

Industrial Occurrence	**Signs And Symptoms**
Bakeries, chemicals production; cleaners; construction; electroplating; engineering; leather; metal; paint; pharmaceuticals; plastics; printing; rubber and textiles etc.	Eczematous lesions.

Diagnosis

1. Occupational history and clinical signs.
   Referring to detailed list of irritants and sensitizers given in the appendix.
2. Observing if many workers in indentical situtation develop cutaneous changes, patch tests.*

* Patch tests may be positive in allergic patients.
* Patch tests need to be done very carefully since **irritant reaction** is possible if concentration is above the critical limit . Patch test are not fool proof **as false negative results are possible**. An **irritant substance** may **sensitise** a person after some period of exposure. On the other hand sensitisers at higher concentrations may cause irritant dermatitis.

c). Skin disease caused by biological agents

Covered in section on Occupational Infectious Diseases (A1).

Hearing impairment caused by noise

Industrial Occurrence	Signs and Symptoms
In industries associated with High noise levels (in textiles, engineering, boilers, explosives, compressors, tanneries etc.)	Four phases of development of the chronic effect: 1. Ringing in the ears at the end of the workshift,slight headache,tiredness and dizziness. 2. Intermittent ringing in the ears. 3. Normal hearing is affected if background noise is present.,incapability of picking up components of a conversation.,cannot hear ticking clock etc. 4. Feeling of hearing insufficiency is manifest. Reduction in hearing capacity is not only quantitative but also qualitative, i.e. sounds are percieved in an abnormal manner e.g. `s', `ch', `th' sounds. Non-auditory effects of noise are: 1. reduces the efficiency of work, 2. pupil of the eye is dilated, 3. sleep disturbances, 4. raised blood pressure,

Diagnosis

1. Occupational history and clinical signs.
2. Monitoring of the work-place.
3. Audiometry - usually 4KHZ level deafness is found primarily, in later stages it affects both lower and higher frequencies.
4. Audiometry has to be repeated after intervals.

Characteristic audiogram:

- Bilateral sensori-neural defect.
- C5 dip at 4000HZ or other `high tone dip

Patient Bandhu Shankar textile worker came to Occupational Health and Safety Centre (OHSC), Mumbai with history of hearing loss noticed first in left ear and later on in right ear. There is no history of high-grade fever suggestive of viral infection, or trauma or use of ototoxic drugs taken systematically or topically.

On examination : Ear - bilaterally tympanic membrane intact and no evidence of infection and nose and throat are normal.

Audiogram shows bilateral severe sensorineural loss.

	Hz 500	Hz 1500	Hz 2000	Hz 3000	Hz 4000
Rt ear db	75	75	70	70	80
Lt ear db	85	100	100	100	100

Diagnosis : Occupational deafness
Disability: 70%

Kindly refer to `Impairments, Disabilities and their Assessment', Published by PRIA, New Delhi

Diseases caused by dinitrophenol, or a homologue, or substituted dinitrophenols or other salts

Examples: 2,3- dinitro phenol; 2,4-dinitro phenol; DNP; etc.

Diseases caused by Dinitrophenol

Industrial Occurrence	Signs and Symptoms
In industries associated with, Chemical production; Dyes; Explosives and Wood preservatives, etc.	**Acute effects** • Poisoning results first in excessive sweating, feeling of warmth with weakness and fatigue is present. • In severe cases there is rapid respiration and tachycardia. Hyperthermia may be fatal.The effects are more severe in hot working places. • Skin becomes yellowish when absorbed through skin. Succeptible individuals may show pruritic dermatitis. If ingested may produce cataract. • Renal effect: Albuminuria. Chronic effects are not recorded.

Diagnosis

1. Occupational history and clinical signs.
2. Derrien's test-urinary excretion of nitrophenol or aminophenol.
3. Renal function test.

Diseases caused by beryllium and its toxic compounds

(Examples: Beryllium chloride; Beryllium fluoride; Beryllium nitrate; Beryllium nitride; Beryllium oxide; Beryllium sulphate hydrate; etc.)

Industrial Occurrence	Signs and Symptoms
In industries associated with Alloys (as hardening agent); beryllium extraction: manufacture of flourescent powders, lamps, and tubes: nuclear reactors (as moderators); steel making (as deoxidizer); use of beryllium ceramics.	**Acute effects** • Nasopharangitis characterised by swollen and hyperaemic mucuous membranes,bleeding points,fissures and ulceration. • Perforation of nasal septum. Following intense exposure,a severe chemical pneumonitis may lead to pulmonary oedema and death. • Skin effects: reddened,vesicular dermatitis after contact with soluble berrylium. **Chronic effects** • Mainly respiratory: begin with feeling of weakness, easy fatigue and weight loss without cough or dyspnoea. • In later stages dry, non-productive cough with exertional dyspnoea may be found. Vital capacity is decreased. • Interstitial lung disease histologically mimicking sarcoidosis and characterised by multiple non - caseating granulomas

	occur. Some granulomas may be found in the liver, spleen and lymph nodes. • Joint pains may occur. • As berryllium is strong irritant,it may produce allergic dermatitis. • Once sensitisation occurs it becomes permanent. • If berryllium enters an abrasion, granulomatous, painless, non-tender swelling may develop. • Conjunctivitis characterised by severe peri-orbital oedema is possible. • Sequelae of toxicity in the form of cor pulmonale, cyanosis, clubbing and CCF. • Renal stones Bronchogenic carcinoma .

Diagnosis

1. Occupational history and clinical signs.
2. Consistent clinical findings.
3. Radiographs show diffuse infiltrates and hilar adenopathy. Infiltrates are of three types- Granular, Linear and Nodular.
4. Impairment of Lung function.
5. Tissue assaay of berryllium.
6. Serum Uric Acid level.
7. In case of involvement of other systems ,relevant specific tests to be done.

Diseases caused by cadmium and its toxic compounds

(Examples: Cadmium chloride; Cadmium oxide; Cadmium stearate; Greenockite or Cadmium sulphide; etc.)

Industrial Occurence	Signs and Symptoms
In industries associated with Alloys for motor cars, aircraft, and marine engines; cadmium-nickel batteries manufacture; easily fusible alloys; electroplating; nickel plating; process engraving; solder for aluminium; yellow, red, orange paints,used in manufacture of: ceramics, glass, leather, plastics, printing inks rubbers, vitreous enamel, etc.	**Acute effects** • Symptoms start after one to eight hours after exposure, with sneezing,cough and irritation of oral and nasal mucousa. • Influenza life manifestations with muscle pain (metal fume fever) are possible. • May cause chemically induced asthma. In severe cases chemical pneumonitis with pulmonary oedema occurs. • Death is possible after 4 to 7 days. • If ingested then features of acute food poisoning occur. **Chronic effects** • Local effect over the respiratory tract may be found in the form of chronic pharyngitis and laryngitis. • Obstructive lung disease in the form of emphysema is the main symptom. • Renal efects - tubular dysfunction • Anemia. • Prolonged or long term exposure leads to proteinuria with increased excretion of calcium and phosphorus and renal stones are frequently found.

	• Bone metabolism may be disturbed. Osteomalacia can be found in chronic exposure. • Essential hypertension • Prostatic Carcinoma. Other effects are loss of weight , loss of appetite and sleep disturbancses.

Diagnosis

1.Occupational history and clinical signs.
2. Blood Cadmium level estimation.
3.Urinary Cadmium level estimation.
4.Tests for renal tubular and glomerular dysfuntion.

Occupational Asthma

Occupational asthma is a disorder initiated or provoked by agents found in the work environment causing asthmatic signs and symptoms either due to excessive concentration of those agents or because of an exaggerated response by the individual worker.
Attack may be

1. immediate within a few minutes or an hour of exposure and may last for a few hours or,
2. delayed and starting upto four hours after exposure and may last for a day or may be repeated on subsequent days without re-exposure.

Industrial Occurrence	**Signs and Symptoms**
In industries associated with Animals and their handling -- nails hair, teeth, feathers; animal debris; cotton; docks; laboratories; metals; pharmaceuticals; plastics; wood; substances in the list of known agents of occupational asthma.	• Bronchial asthma is characterised by paroxysms of dyspnoea accompanied by wheezing. • In episodic asthma paroxyms of wheeze and dyspnoea occur at any hour of the day or night, or sudden onset and may be preceded by a feeling of tightness in the chest. Breathing is exhausting and initially expiration is difficult and prolonged, compared to inspiration. • The wheeze is chiefly expiratory and there is often an unproductive cough which aggravates the dyspnoea. • Recurrent attack of Upper Respiratory Tract infection is common. • Reversibility of symptoms is lost on long standing exposure. • There is progressive destruction of the lungs.

	• Sudden death can occur due to occupational asthma.

Diagnosis

1. Occupational history and clinical signs.
2. Clinical examination.
3. Serial peakflow testing for reduction in the peakflow is a very specific test for screening patients.
4. Bronchoprovocative test can be dangerous and can have false negative.
5. Routine blood examination may show eosinophilia.
6. Sputum may contain eosinophils.
7. Allergen test on the skin (may be false negative).
8. Lung function test shows characterstic changes.
9. If the symptoms subside when away from work and are aggravated after starting work, it is a strong indication.

Known agents of occupational asthma :

Abietic acid, acacia, acacia gum, acaridae, acrylic fibres, acrylic-precursors, actinomyces, alicyclic amines, aliphatic aldehydes, aliphatic amines, alkaline persulphates, alkyl phosphates, ammonium chloroplatinate, ampicillin, anthraquinone dyestuffs, antibiotics, arabic gums, aromatic amines, anthropods, azo dyes; Bacillus subtilis, barley, benzalkonium chloride, benzyl penicillin, betalactamines, bromelin;

Candida tropicalis (proteins), carbamates, castor oil, cats, chlorine, chlorthion. chromium, cobalt, cockroaches, coffee-green, colophony, cotton, cows;
D D V P, detergents enzymatic, diazinon, diazomethane, diethanolamine, diethylene diamine, diethylene triamine, dogs; Enzymatic detergents, epoxy resins and hardeners, ethyl hexamine, exotic woods;
Flax, flour or meal, formaldehyde;

Grain dusts, graminaceous pollens, green coffee, groundnuts, guinea pigs;
Hair, horns, feathers, etc., hamsters, henna, hexamethylenetetramine;
Industrial perfumes, insecticides, ipecacuanha;
Jute;
Karaya gum;
Laboratory animals, lead liquorice, locusts;
Maleic anhydride, metampicillin, methylene bisphenyl isocyanate, methyl isocyanate, mercury (organic compounds), mice, mites, moulds;
Nickel, nitric oxide;
Oats, oil cake, oleandomycin, organic isocyanates, organo-phosphorus compounds;
p-dichlorobenzene, p-formaldehyde, p-phenyldiamine, panonychus ulmi, papain, penicillins, persulphates, pesticides, phenyl-formaldehyde resins, phenylglycine, phenylhydrazine, phenylmercuricnitropropionate, phosphoramines, phthalic acid, phthalic anhydride, piperazine, platinum salts, polyamides, polyesters, polyvinyl chloride, proteolytic enzymes, pyrethrum;
Quinine;
Rabbits, rats, red spiders, rice dusts, rye;
Sericin, silk, soya, spiramycin;
Textiles natural, textiles synthetic, thrombin, triethylene diamine, triethylene tetramine, trimellitic anhydrides, tungsten carbide;
Urea-formaldehyde resins;
Vanadium, vanilin, vegetables, viscose;
Wasp, bite, water fleas, welding fumes, wood, wood pulp dust, wool;

Diseases caused by fluorine and its toxic compounds

(Examples: Aluminium fluoride; Cryolite or sodium-aluminium fluoride; Fluorite or fluorspar, calcium fluoride; Freon-11, Freon-12 and other fluorocarbons; Salts of hydrofluoric acid; Sodium fluoro aluminate; etc.)

(a)Flourine

Industrial Occurence	**Signs and Symptoms**
In industries associated with Aircraft piston engines; conversion of uranium tetrafluoride to uranium hexafluoride; electrolyte refining and pickling of metals; electroplating; etching of glassware; manufacture of: artificial cryolite, and certain other refrigerants; high-octane petrol synthesis by alkylation (as hydrofluoric acid);	**Acute effects** May cause strong irritation of the mucous membrane of the aero-digestive tract. Pulmonary oedema may develop. Even respiratory paralysis and death are possible. • Haematemesis, abdominal pain and diarrhoea, convulsion, coma and death may occur. **Chronic effects** • Damage of respiratory tract and kidneys may occur. • Occasionally pulmonary fibrosis may develop. • Fluorosis of bones and ligaments characterised by increased radiographic bone opacity. • Formation of blunt excrescences on the ribs, calcification of intervertebral

insecticides; metallic aluminium (for aluminium fluoride intermediate). Chemical weapons; insecticides; pesticides; rat poison. Etching glass; fluorides; fluorocarbons and refining of metals.	ligaments and formation of bony spicules over the tibia, fibula and pelvic bones and spinal column. Calcification of ligaments, polyarthralgia are possible. • Restriction of movement and tendency towards fracture are reported but not documented. Deformity of bones may occur.

Diagnosis

1. Occupational history and clinical signs.
2. Skeletal survey.
3. Urinary excretion levels of fluorides.
4. Lung function test.

(b) Diseases caused by Flouroacetic acid and its compounds,sodium-flouroacetate etc.

Industrial Occurrence	**Signs and Symptoms**
In industries associated with Rodenticide and mamalian pesticide; used in chemical warfare.	**Acute effects** • Nausea, vomiting, excessive salivation, pain in the stomach, twitching of muscles, apprehension and low blood pressure. • After around 6 hours; convulsions, coma depression. • Death may result due to asphyxiation during coma, ventricular fibrillation and cardiac arrest.

	• Major toxic effects are on central nervous system and cardiovascular system.

Diagnosis

1. Occupational history and clinical signs.
2. Chemical analysis - Organically bound fluorine in body is most reliable proof of poisoning.
3. Increase of citrate in urine.

(c) Hydrofluoric Acid

Industrial Occurrence	**Signs and Symptoms**
In industries associated with Used as catalyst for high octane gasoline; in solutions for frosting, etching, polishing glass, pottery production and pickling of metals; used for removing sand from metal castings, production of fluorides, flurocarbons and freons.	**Acute effects** • Local Burns • Erythema • Pain after 1 to 2 hours • Oedema • Blistering after 24 hours • grey areas of necrosis deep ulceration **Later :** • tenosynovitis, • osteolysis. **Systemic effects** • Nausea. • Abdominal colic. • Muscular pains. • Convulsions. • Paralysis.

- Hypertension.
- Cardiac arrythmias.
- Cardiac failure.

Eye

- Corneal abrasion
- Conjunctival injection.
- Chemosis
- Aphakia, destruction of lens and eye structure.

Inhalational injuries

- Haemoptysis
- Pulmonary oedema
- Respiratory obstruction

Chronic effects

- Corneal erosions.
- Keratoconjuntivitis sicca.
- Progressive vascularization of cornea.
- Symblepheron
- Altered pulmonary functions(reactive airway dysfunction syndrome).
- Flourosis(refer to section on **Flourine**)

Diagnosis

1. Occupational history and clinical signs.
2. Fluoride concentration in urine.
3. Lung function test.
4. Skeletal survey.

Diseases caused by nitroglycerine or nitroacid esters

(Examples: Nitroglycerine; nitrocellulose; nitrocellulose acetate; etc.)

Industrial Occurence	Signs and symptoms
In industries associated with manufacture of cardiovascular drugs; explosives.	• Mainly hypotensive action following dilatation of arteries characterised by increased heart rate, fall of blood and pulse pressure. • Adaptation to the hypotensive action is seen among the workers with nitroglycerine but discontinuation of exposure may interrupt this adaptation. • A period of nausea when resuming work on monday morning is seen. Week end angina - patient may die suddenly at the end of the week-end or vacation due to angina and its after effects. **Acute Effects** The initial symptom of exposure are headache, dullness and reduced blood pressure followed by nausea, vomiting with consequent fatigue and weight loss and cyanosis, possibly hallucinations.

	Chronic Effects • Prolonged exposure may result in neurological disorder. • Manifestation is like acute mania. • In severe poisoning, confusion, hallucinations and maniacal manifestations have been observed. • Tremors and neuralgia. • There are gastro intestinal disturbances. • Skin irritation and erruptions on the palms and interdigital spaces and ulcers under the nails have been observed. • Tubular interstitial nephritis is possible and may lead to acute renal insufficiency.

Diagnosis

1. Occupational history and clinical signs.
2. Pulse rate and blood pressure.
3. Electrocardiogram.
4. Urine examination for tubular dysfunction.
5. Plethysmography.

Diseases caused by alcohols and ketones

(Examples of alcohols: 2-Ethyl hexanol; Ally alcohol; Amyl alcohol or 1-Pentanol, primary amyl alcohol; Cyclo hexanol; Ethyl alcohol or ethanol; Furfuryl alcohol or 2-furi carbinol, furfural alcohol; Isoamyl alcohol; Isobutyl alcohol or 2-butanol; Isopropyl alcohol or 2-propanol; Methyl alcohol or methanol; n-butyl alcohol or 1-butanol; n-propyl alcohol or 1-propanol; etc.)

a) Ethyl alcohol (Ethanol)

Industrial occurrence	**Signs and Symptoms**
In industries associated with Production of acetaldehyde; butadiene (used in plastic and synthetic rubber); chloroethane, (as a solvent); cosmetics; drugs; lacquers; perfumes; plasticisers; plastics; polishes ; rubber accelerators.	**Acute effects** • Irritation of eyes and mucous membranes, headache, drowsiness, fatigue. • Central nervous system depression • It dissolves fat in the skin and causes dermatitis. • Splash in the eyes causes immediate burning • Workers with damaged liver are more affected **Chronic effects** Central nervous system depression, headache.

Diagnosis

1. Occupational history and clinical signs.
2. Signs of central nervous system depression, and irritation of eye and upper respiratory tract.

b) Methanol (methyl alcohol)

Industrial occurrence	**Signs and Symptoms**
In industries associated with Production of antifreeze mixtures; cement; coated fabrics; dewaxing preparations; dyes; embalming fluids; ethylene glycol; formaldehyde; inks, methacrylates; methyl amines; methyl halides; paints, photographic films; plastics; resins; textile soaps; unshatterable glass; waterproofing formulations (as solvent) adhesives; and dyes.	• It can enter the body through inhalation, by mouth, and can be absorbed through skin. • Death may occur even due to absorption through skin. • If swallowed or inhaled in large amounts, it can cause blindness. **Acute effects** • Irritation of mucous membranes, headache, ringing in ears, fainting, vomiting, colic, insomnia, dilated pupils, constant movement of eyeballs (nystagmus) and skin injuries on hands, wrists, and forearms. • Metabolic acidosis with associated complications like delirium, stupor, coma and even death due to respiratory failure. **Chronic combined exposure to methyl alcohol and carbon monoxide has been reported as a causative factor of thickening of walls of arteries pertaining to the brain.**

Diagnosis

1. Occupational history and clinical signs.
2. Ophthalmological examination
3. Presence of formic acid in urine. Also suggested is the measurement of methylalcohol in urine at the end of workshift.
4. Relevent tests for metabolic acidosis.

(c) Glycols : Ethelyne Glycol

Industrial Occurrence	**Signs and Symptoms**
In industries associated with Used for depression of freezing point, lubrication, solublisation, in production of explosives, resins, cosmetics, and additives to food; as chemical intermediate.	**Acute effects** • Central nervous system depression, nausea, vomiting and abdominal pain are early manifestation of acute poisoning. • Subsequently renal failure with oliguria, protinuria and large amounts of oxalate crystals in the urinary sediment. • Metabolic Acidosis

Diagnosis

1. Occupational history and clinical signs.
2. Urine examination for oxalate crystals.
3. Relevant tests for metabolic acidosis.

d) 2-Propanol

Industrial Occurrence	**Signs and Symptoms**
In industries associated with Used as solvent in pharmaceutical, household and personal products, used in the	**Acute effects** Central nervous system depression, twice as active as ethanol in comparable doses Respiratory depression Irritation of eyes Hyperglycaemia Increase CSF proteins

production of acetone, denaturant, coolant in beer manufacture, dehydrating agent, de-icing agent, flavouring agent, polymerization modifier	Atelectasis **Chronic effects** 1. Itching eczematoised dermatitis 2. Cancer of paranasal sinuses 3. Laryngeal cancer

Diagnosis

1. Occupational history and clinical signs.
2. Acetone in blood/urine.
3. Blood sugar level.

(e) Diseases caused by acetone

Industrial occurrence	**Signs and Symptoms**
In industries associated with Production of acetic anhydride; chloroform; diacetone alcohol; iodoform; Vitamin C (as solvent); acetylene; cellulose; collodion; cotton; fats; lacquers; oils;	**Acute effects** • Irritation of skin and mucous membranes. • Exposure to high concentrations leads to stupor, diffuculty in breathing and unconsciousness and collapse. • Possible kidney and liver damage. **Chronic effects** • Repeated exposure may lead to headache. • General weakness accompanied by blood

(used in production of) celluloid; dyes; explosives; lacquer; leather (synthetic); lubricants; rubber; silk (artificial), varnish.	changes like increase of leucocyte and eosinophil count. • Prolonged contact with skin causes dryness and diffuse redness.

Diagnosis

1. Occupational history and clinical signs.
2. Albumin, and red and white blood cells in urine indicate damage to kidneys.
3. High levels of urobilin and bilirubin indicate damage to liver.

(f) Diseases caused by other ketones

Industrial Occurence	**Signs and Symptoms**
In industries associated with Production of, artificial silk; cosmetics; explosives; perfumes; pharmaceuticals; plastics (as solvent); dyes; fats; gums and resins.	**Acute effects** • Ketones are narcotic when inhaled in high concentrations. • At lower concentrations they are irritating to eyes, skin and respiratory system. • Excessive exposure leads to the central nervous system depression. • Sensory and motor nerves are affected. Methyl butyl ketone (MBK) may cause motor and sensory neuropathy.

Diagnosis

1. Occupational history and clinical signs.
2. Periodic medical examination with attention to nerve conduction velocity, the central nervous system, the respiratory system, eyes, renal system and liver.

Diseases caused by asphyxiants: carbon monoxide and its toxic derivatives, hydrogen sulphide

(a) Diseases caused by Carbon monoxide

Industrial Occurrence	Signs and Symptoms
In industries associated with blast furnaces; boilers; garages; industrial gases; metallurgy (as reducing agent); mines; organic syntheses; production of metal carbonyls and tunnel construction and maintenance.	**Acute effects** • Headache, abnormal rapidity of respiration, nausea, weakness, dizziness, mental confusion, hallucinations, slightly bluish, grayish, slate-like or dark purple discoloration of the skin (cyanosis) due to chemical reduction of haemoglobin in blood, temporary loss of consciousness due to reduced oxygen supply to the brain. • Acute exposure may lead to death. • Impaired visual acuity and dark adaptation • Even after signs of improvement mental damage may remain. **Chronic effects** • Chronic behavioural changes • Longer Exposure may cause aggravation of angina pectoris and other cardio vascular disorders. • Chronic combined exposure to methyl alcohol and carbon monoxide has been reported as a causative factor of thickening of wall of arteries pertaining to the brain. • Fetal abnormalities.

Diagnosis

1. Occupational history and clinical signs.
2. Blood cherry pink in color.
3. Level of carboxyhaemoglobin.
4. Electrocardiographic tracings.

(b) Diseases caused by phosgene (toxic derivative of carbon monoxide)

Industrial occurrence	**Signs and symptoms**
In industries associated with Insecticides production; metallurgy; pharmaceuticals (industrial syntheses such as manufacture of) acid chlorides; carbonic acid esters; coal tar; dye stuffs; isocyanates and their derivatives; urea. Pesticide production (carbamates)-- used as intermediate; pharmaceuticals.	**Acute effects** • Eye irritation, dryness or burning sensation of the throat, vomiting, pain in chest, cyanosis. • Severe skin or eye burns due to splashes of liquefied phosgene • The symptoms of severe respiratory distress may be delayed upto 72 hours. • Delayed onset of pulmonary oedema with cough and foamy sputum, severe cyanosis, may lead to pneumonia. Death is possible due to cardiac failure.

Diagnosis

1. Occupational history and clinical signs
2. Electrocardiogram
3. Chest X-ray

(c) Diseases caused by hydrogen sulphide

Industrial occurrence	Signs and symptoms
In industries associated with Agriculture (as disinfectant); organic sulphur compounds such as thiophene; present around sewers, oil wells and where petroleum products are processed, stored or used; production of inorganic sulphides; sulphuric acid. * By-product in many processes; * Also results from natural decay of organic matter.	**Acute effects** • Above 50 ppm it causes olfactory fatigue. • Prolonged exposure to 500 ppm causes inflammation of nose, pharynx, bronchi and lungs. • Sudden death may happen due to respiratory paralysis or coma. • Possibility of conjunctivitis with pain, watering of eyes, and photophobia. This can progress to blisters on cornea . • Oedema of lungs is possible **Chronic effects** • Neurological deficits • Anxiety • Poor memory • Decreased libido • Dysarthria • Neuritis of acoustic nerve. • Working in environment with 10 ppm for many days may cause headache, conjunctivitis, digestive disorders, weight loss, weakness and problems in breathing. • Anginal pain • Headache • There are reports about nervous system disorders such as paralysis, meningitis, and psychological disorder.

Diagnosis

1. Occupational history and clinical signs.

(d) Diseases caused by Acetonitrile

Industrial occurrence	Signs and Symptoms
In industries associated with Extraction of vegetable and animal oils; Solvents ; used as a chemical intermediate.	**Acute effects** • Suffocation, death is possible. • Nausea, vomiting, chest pain, stupor, convulsions. • May cause eye, skin irritation. • Haemoptysis • Respiratory failure **Chronic effects** • Headache. • Weakness. • Changes in taste and smell. • Irritation of throat. • Vomiting. • Exertional dyspnoea. • Lachrymation. • Abdominal colic. • Precordial pain. • Disturbances in accommodation. • Salivation. • Psychotic episodes. • Goitre.

Diagnosis

1. Occupational history and clinical signs.
2. Measurement of blood pH, plasma bicarbonate and blood lactic acid.
3. Levels of cyanide in blood.

(e) Diseases caused by Acrylonitrile

Industrial Occurrence	Signs and Symptoms
Industries associated with Production of polymers and co polymers for synthetic fibres, resins, plastics and nitrile rubber industries; used for organic syntheses and as pesticide fumigant.	**Acute effects** • Dull headaches. • Fullness of chest. • Irritation of ear, nose and throat. • Itching of skin. • Nervous irritability. • Vertigo. • Nausea. • Convulsions. • Jaundice. • Dermatitis. • Haematological changes seen. • May be fatal. **Chronic effects** Cancer of 1. lung 2. livergall 3. bladder

Diagnosis

1. Occupational history and clinical signs.
2. Measurement of blood pH, plasma bicarbonate and blood lactic acid.
3. Urine test for acrylonitrile metrapturic acid.

(f) Diseases caused by asphyxiants due to being respiratory irritants

Respiratory irritants

(i) Diseases caused by acids: Hydrochloric acid (HCl) , Nitric acid ($HNO_{3)}$ and Sulphuric acid (H_2SO_4).

Industrial Occurrence	**Signs and Symptoms**
HCl *In industries associated with* Acidizing oil wells, artificial silk, chlorinated chemicals, dyes, dye intermediates, photographic industry, pickling of metals, pigments, refining of edible oils, refining of ores, refining of soap, rubber, textile. **HNO_3** Cleaning of metals, *Production of* acqua regia, arsenic acid, dyes, dye stuffs, explosives, fertilisers, jewellery, metalic nitrates,	**Acute effects** **Skin** • Pain • Erythema • Oedema • Coagulative necrosis. • **Inhalational** • Pharyngitis • Laryngitis • Rhinitis • Epistaxis • Haemoptysis • Pulmonary oedema **Chronic effects** 1. Bleaching, discoloration of skin 2. Chronic dermatosis 3. Nasal septum perforation 4. Erosion of enamel of incisor teeth 5. Dyspnoea on exertion and reactive airway dysfunction syndrome (RADS)` with decrease in FEV_1 , and FVC.

pharmeceuticals, sulphuric acid. **H_2SO_4** *Production of* Chemicals, explosives, fertilisers, glucose, hudrochloric acid, metal cleaning, oleum, pharmaceuticals, phosphoric acid, plastics, super phosphates, pickling of metals, *Refining of* mineral oils, vegetable oils, petroleum *Used in* laboratories	RADS is caused by a single exposure to any of the respiratory irritants. This has a distinctly different pathogenesis from occupational asthma (which occurs only in allergic individuals) but symptoms are similar and characterised by recurrent attacks.

(ii)Ammonia

Industrial occurrence	**Signs and Symptoms**
Industries associated with refrigeration, petroleum, refining; used in manufacture of fertilizers, nitric acid, explosives,	**Acute effects** 1. Burning sensation in eyes, nose, throat. 2. Respiratory distress. 3. Tachypnoea. 4. Cough. 5. Laryngeal oedema. 6. Pulmonary oedema.

plastics and other chemicals.	7. Bronchopneumonia. 8. Lacrymation. After oral exposure 1. Caustic burns of oesophagus/stomach. 2. Supraglottic oedema. 3. Fatality possible. 4. Neurological syndrome: irritability, tremors, hyper reflexia, delirium and coma. **Chronic effects after oral exposure** 1. Chronic bronchitis. 2. Bronchiectesis. **3.** Oesophageal stricture.

Diagnosis

1. Occupational history and clinical signs.
2. Blood ammonia level.
3. Blood urea nitrogen.
4. Urinary urea nitrogen.
5. Urinary ammonium levels.

(iii)Chlorine

Industrial occurrence	**Signs and Symptoms**
In industries associated with metal fluxing; used in sterilization of water supplies and swimming pools; bleaching agent; reagent in synthetic	**Acute effects** Asphyxiating phase • Burning sensation in the throat. • Cough. • Dyspnoea. • Aphonia. • Bradycardia.

chemistry; detinning and de-zincing iron.	• Cyanosis. • Hypothermia. Post-asphyxiating phase • Severe bronchitis. • Pulmonary oedema. **Chronic effects** • Irritable heart. • Neurosis. • Bronchitis. • Asthma. • Olfactory deficiency.

Diagnosis

1. Occupational history and clinical signs.
2. Lungs function test can help

Lung Cancer and Mesotheliomas caused by asbestos

This head applies when effects of exposure lead to lung cancer and mesotheliomas.

For description of general effect of exposure to asbestos and resulting diseases, please refer to `Asbestosis' , C-1(b), page 99.

Primary neoplasm of the epithelial lining of the urinary bladder or the kidney or the ureter

Industrial Occurence	Signs and Symptoms
In industries associated with Auramine; Benzidine; dyes; hair dyes; herbicides; magenta dyes (like fuschin, rosaniline); naphthylamine; printing inks; rubber; textile dyeing and textile production.	• Major sign is painless haematuria. • Haematuria may last for a few hours or a few days before it stops. After a few weeks or months, bleeding starts again. • Intervals between two periods of haematuria become shorter, haematuria becomes severer and of longer duration. • In case of cancerous tumors, haematuria eventually becomes continuous. Urinating may become painful. • Pain in front of thighs. • Colic is felt from back to front. • Dragging pain in loins is a sign of tumor in kidneys, flank pain, lower limb oedema, and pelvic mass. • Occasionally primary growth remains silent. Complaints of bone swelling or spontaneous fracture, suggestive of metastasis may be present. • In later stage haematuria becomes continuous and difficulty in passing urine develops.

Diagnosis

1. Occupational history and clinical signs.
2. Urine analysis.
3. Cystoscopy and biopsy.
4. Intravenous pyclography (IVP).
5. CT scan of kidney, ureter and bladder.

Pneumoconiosis caused by sclerogenic mineral dusts

The diease pneumoconiosis is the generic description of diseases of the respiratory tract caused by inhalation of dusts. Only the sclerogenic mineral dust induced diseases are covered by the existing law.

(a) Silicosis and Silico-Tuberculosis

Silicosis results from inhalation of any of the three forms of silicon dioxide; quartz, cristobalite and tridymite.

Industrial Occurence	**Signs and Symptoms**
In industries associated with Certain foundry operations like sandblasting, etc.; cutting of quartzite; agate; gneiss, granite and slate; knife scissors sharpening with grinding stone; manufacture, handling and transport of cement; mines and quarries; sculpture from stone; stripping and refining of steel furnaces;	(i) **Acute silicosis** - Occurs due to high level exposure to dust. • Patient gets dry cough and breathlessnes which starts within a few weeks of the start of exposure. • The symptoms worsen gradually despite treatment. Chest X-ray - Shows picture of patchy pulmonary oedema, which gradually hardens and the affected area shrinks. Pulmonary function - Shows findings of restrictive lung pathology. There is no treatment for acute silicosis or any other form of silicosis. This type is often fatal, depending on the amount of dust retained in the system before stoppage of exposure.

(manufacture of) glass, porcelain; pottery; slate pencil.	(ii) **Chronic Silicosis** : This is actually the same disease as above, caused due to lower dose of exposure or exposure to lesser toxicity of the dust, for a prolonged period. Here the worker gets symptoms only after the disease has progressed. Symptoms being :- 1. Difficulty in breathing, occassionally just mild breathlessness on exertion. 2. Features of acute respiratory infection. 3. Features of cor-pulmonale in late cases. 4. There may be symptoms and signs of tuberculosis along with/without any of the above.

Diagnosis

1. Occupational History and clinical signs.
2. Chest X-ray, which can show, Nodular or/and linear, shadows, predominantly in upper zones, can also be evenly distributed. There can be large opacities also. [pulmonary massive fibrosis (PMF)]

- Hilar node enlargement, occassionally 'egg shell calcification'.
- Pulmonary function is necessary for assessment of functional disability. Shows restrictive lung pathology. For regular screening of workers FVC and FEV_1 are to be done along with chest X-rays.

(iii) Silico Tuberculosis

Risk of developing tuberculosis after one has got silicosis is 15 times more than the general population.

It can well be so, that an asymptomatic silicosis patient turns up early as he has contracted tuberculosis and has cough, sputum, loss of weight etc.

Investigations may show :-
Chest X-ray to be having - Soft shadows of tubercular breakdown with pre-existing silicotic opacities. Sputum for AFB can be positive.

Looking the other way round all tuberculosis patients having a definite occupational history of "dusty" jobs of some years duration should be screened for early or not so early evidence of pneumoconiosis.

Even there may be cavitary tuberculosis with haemoptysis as the presenting features in cases of PMF or confluent sillicotic nodules.

b) Asbestosis & Asbestos Related Diseases of Lungs And Pleura

Industrial occurrence	**Signs and Symptoms**
In industries associated with Brake linings, cement, filler for plastics, fire smothering blankets, mining of asbestos, safety garments, thermal and electric insulation.	Asbestosis gives rise to no specific symptoms apart from 1. Very gradually increasing breathlessness. 2. Cough and Sputum only in very advanced stage. 3. Tightness in chest and inability to breath in full (not associated with pleuritic or angina like pain). Patient may also come with benign pleural effusion or can be a chance diagnosed based` on chest X-ray due to detection of pleural plaques. **In asbestosis there can be four manifestations of the disease which can co-exist** I. **Pulmonary fibrosis** - Characterised by • Increasing dyspnoea. • Cough . • Clubbing. • End inspiratory and early expiratory crepitations at the bases of the lungs. • Chest X-ray will show bilateral irregular opacities of lower zones. • Combination of pleural and parenchymal shadows produces haze of cardiac margin (shaggy heart). • Progessively diminishing FVC and increase in FEV_1/FVC ratio.

II. **Benign pleural effusion** - Common in young and middle aged workers, the disease is not dose related. The period of exposure being for 1 to 2 years only.

There may be

- Influenza like febrile illness followed by discomfort on one side of chest and breathlessness in otherwise fit person.
- Chest X-ray showing pleural effusion on one side.
- Pleural fluid may be blood stained.
- Raised ESR, leucocytosis with relative lymphocytosis.
- Effusion may subside with or without cortico-steroid therapy.
- There may be recurrence on either side.
- There may be development of diffuse pleural thickening.

III. **Benign pleural Plaques**

Workers with moderate to heavy exposure to the offending dust can present with

- Obliteration of costophrenic angle on X-ray.
- Thickening of diaphragmatic pleura, which may extend upwards to parietal pleura.
- Thickened pleura may become calcified.
- There can be co-existent fibrotic lesions of asbestosis with full blown features.

Diagnosis

1. Occupational history and clinical signs.
2. Chest X-Ray.

(iv) Mesothelioma of the pleura and peritonium

It is a lethal disease which can occur even from a relatively trivial exposure.

Mesothelioma has got no other identifiable aetiological cause except asbestos exposure.

Signs and Symptoms

Patient initially presents with

- Breathlessness of sudden/gradual onset
- There may be discomfort or heaviness of one side of the chest
- There may be patchy changes in skin sensation and sweating
- There is usually no appreciable deterioration of health.

There are signs of

- Pleural effusion, often blood stained
- Conspicously the medistinum shifts to the affected side with crowding of ribs in the early stage of the disease which later on can be reversed.

Diagnosis

1. Occupational history and clinical signs.
2. Pleural biopsy and aspiration for cytology
3. X-ray appearence of irregular parietal wall tumor and exclusion of bronchial cancer
4. CT scan

Bronchogenic Carcinoma, related to asbestosis may be possible.

(c)Anhtraco-Silicosis or Coal Workers' Pneumoconiosis

Industrial Occurrence	Signs and Symptoms
In industries associated with Coal mines, coal handling in any industry.	In case of Anthraco-silicosis or coal miners pneumoconiosis, symptoms develop late, when the disease has progressed. So routine periodic X-ray of workers exposed to risk is the only mode to detect the disease early and take any measures to contain the destruction. Radiologically the disease is classified as :- **(i). Simple Coal Workers' Pneumoconiosis (C.W.P.) :** The grading is done by matching Chest X-ray plates with standard ILO plates for comparative size of rounded opacities and irregular opacities and the extent of their spread in the X-ray. (A standard practice in all forms of Pneumoconiosis) Symptoms are absent in early cases, in long standing cases there is - 1. Progressive breathlessness on exertion subsequently even at rest. 2. Cough, sputum, may be black and melanoptysis is possible. 3. Pulmonary function shows impairment. 4. There may be signs of Chronic obstructive pulmonary disease (C.O.P.D) and cor- pulmonale with or without congestive cardiac failure (CCF).

	ii. Progressive Massive Fibrosis (PMF): This type is radiologically characterised by large (3 to 10 cm long), one or multiple, unilateral or bilateral opacities, usually in upper or mid zone. The opacities may occasionally be irregular in shape, may cavitate. The disease is occasionally complicated by tuberculosis. It is progressive in nature. Symptoms are the same as in case of simple coal workers pneumoconiosis, but there is progressive deterioration of the patient.

Diagnosis

1. Occupational history and clinical signs.
2. Chest X-ray (comparison with standard ILO plates).

Coal mine workers are also affected due to Chronic Obstructive Lung Disease (COLD) as reported by Dr. Saiyyed H.N , et al.

Bagassosis

Bagasse is the fibrous cellulose residue of sugar. Inhalation of bagasse dust which contains thermophilic microbe "Thermoactinomyces Sacchari" causes Bagassosis, actually a form of extrinsic alveolitis (EAA).

Industrial Occurence	**Signs and Symptoms**
In industries associated with Building materials (insulating wall board, fillers for veneered doors); cardboard; chemically treated pressboards; explosives; fertiliser; fuel; paper; poultry feed; refractory bricks.	**Like other forms of EAA, Bagassosis also is present in two forms:** **Acute effects** Where high dose exposure occurs. Symptoms start several hours after exposure. • Influenza like symptoms. • Breathlessness develops and cough with sputum and even haemoptysis. • There may be constitutional upset, headache and muscle pain. • There may be severe loss of appetite and weightloss. • If exposure is stopped, the symptoms usually abate in two to ten days. • If exposure continues the symptoms increase and there appears inspiratory crackles on auscultation. • Chest radiograph may show generalised haze with loss of sharpness of vascular markings. There may be coalesced

	nodules appearing as patchy consolidation. • Pulmonary function test shows obstructive and subsequently restrictive changes.
	Chronic effects This form differs from acute illness by the appearance of irreversible pulmonary fibrosis which usually manifests as having the history of exposure for two to three years or longer with - • Usually progressive breathlessness. • Considerable weight loss. • Cough, sputum. • Scattered inspiratory crackles in lung fields. • Chest X-ray shows chronic irreversible changes such as linear shadows, honey combings, collapse of lobe (usually upper) with compensatory emphysema of lobes(usually lower). • Pulmonary function test shows restrictive changes .

Diagnosis

1. Occupational history and clinical signs.
   and any one of the following .
2. Partial or total reversibility of symptoms in initial stages on withdrawl and reexposure.
3. Charactersitc chest X-ray or Lung function changes.
4. Demonstration of precipitating antibodies (precipitins) to the causal antigen in patient's serum.

Byssinosis

This is a disease caused by the inhalation of cotton, flex, hemp and sisal dust. Usual duration of exposure before initial symptoms appear is about five years.

Industrial Occurrence	Signs and Symptoms
In industries associated with Bale pressing plants; Cotton, Flax; Gins; Linen; Mixing and card rooms; Rope making; Soft hemp; Textiles; Twine making.	• Acute symptoms of chest tightness and breathlessness charaterstically developing on the first day of the working week, usually over second half of the workshift. • Symptoms worsen on exertion . • Some may have very rapid onset of chest tightness within an hour of starting work on the first day of the week. • As the disease progresses chest tightness and/or breathlessness extends to the other days of the week. • Eventually, the workers may become severely affected on every working day with permanent and severe effort intolerance.

Diagnosis

1. Occupational history and clinical signs.
2. Characterstic clinical findings .
3. Lung function test.
4. Patient having productive cough on waking up in the morning for more than three months in a year for consecutive three

years and having byssinosis is called atypical byssinotic or byssinotic with chronic bronchitis.

5. Radiology does not help in the diagnosis.
6. Arterial blood gas analysis does not help in diagnosis.

Case history of the first compensated Byssinosis in Bombay

Sampat Tapare, age 42 years, non-smoker working in the weaving department of Mumbai Textile Mills for 25 years came to the Occupational Health and Safety Centre Bombay with chief complaints of

1. *Exertional dyspnoea Gr. III*
2. *Chest tightedness*
3. *Parasternal chest pain aggravated by cough*
4. *Productive cough on waking up in the morning for more than 3 months in a year*

All the symptoms were aggravated following exposure to cotton dust and has it on all days of the week. Many years back he had symptoms only on joining work after a weekly off. He had pulmonary T.B. for which he had taken a complete treatement for 9 months.

On examination, he had bilateral Rhonchi

On investigations : PFT showed FVC : 1.42 L
FEV_1 : 1.21 L
FVC / FEV_1 : 85%

The FEV_1, was 59% of the expected FEV_1 for Indian males aged 42 years.

Diagnosis : Byssinosis
Disability: 60%

Kindly refer to `Impairments, Disabilities and their assessment', Published by PRIA, New Delhi.

Extrinsic allergic alveolitis caused by inhalation of organic dusts

Industrial Occurrence	**Signs and Symptoms**
In industries associated with Animal and vegetable matter processing.	**Acute effects** • Breathlessness, dry cough. • Sometimes fever, aches and muscle pains • Usually after four to ten hours of exposure, crackling sound on auscultation • Symptoms persist for about a day or two if no further exposure. **Chronic effects** With repeated exposures breathlessness becomes continuous and progressive without wheeze.

Diagnosis

Either 1,2,4 or 1,3,5 below are sufficient

1. Occupational history with detection of antigen in work environment
2. Appreance of symptoms of acute effects with lag of about four to ten hours after exposure
3. Demonstration of antibodies (precipitins) to the causal antigen in patients serum.
4. Fine nodular or reticulo nodular shadows or /and fibrotic changes in chest X-Ray
5. Charactersitic changes in pulmonary functions.

Broncho-pulmonary diseases caused by hard metals (i.e., metal carbides)

Example : Cobalt Carbide

Industrial Occurence	**Signs and Symptoms**
In industries associated with Metal carbides, (cemented or sintered) -- mixing, screening, grinding and reshaping of these. Inhalation of dust of metal carbides (cutting edges of machine tools) can cause the following disease.	• Work related asthma which subsides during holidays. • Dry/wet bronchitis with or without broncho- spasm • Difficulty in breathing. • Occassionally pneumonitis. • Interstitial fibrosis, usually following atleast one or the other manifestations above. The disease is severe in case of cobalt carbide. It causes pulmonary oedema and diseases of heart muscles.

Diagnosis

1. Occupational history and clinical signs.
2. Radiological changes
3. Diffused finely striated increased lung markings
4. Soft, patchy, nodular opacities extending usually over the entire lung field with or without thickened hilar shadow.

Biological Monitoring

The tests are advised to be done at the end of the work shifts. The figures given are more useful to detect the effects at early stages and also are useful for judging correctness of treatment.
Absence of elevated levels should not be taken as an absolute basis for denying the diagnosis of an occupational disease.
The response to exposure to a chemical depends on inherited and acquired characteristics and the life style of the human subject and the circumstances of contact.
The levels indicate when none exposed persons may show relevant symptoms. In some individuals the symptoms may occur even before the levels indicated here below are reached in the body.

Lead

Adults	<40 micro gm/ 100ml lead in blood
Reproductive age	<30 micro gm/ 100 ml lead in blood.
Children	<10 micro gm/ 100 ml lead in blood.
ALA	<2 mg/100 ml in urine
Coproporphyrin	<15 micro gm/ 100 ml in urine
Tetraethyl lead	>35 micro gm/ 100 ml lead in blood with <50 micro gm/ 100 lead in urine
Nitrous fumes	Methemoglobin less than 1% of haemoglobin.
Organophosphurus Compounds	More than 20% depletion of the exposed person's normal cholrinesterase level.
Mercury	> 1 micro gm./ 100 ml mercury in blood indicates increased absorption. should not exceed 3 micro gm/ 100 ml in blood. should not exceed 5 micro gm /100 ml in urine.

Benzene	Phenol in urine < 1 mg /100 ml or 50 mg per gm of creatinine.
Nitrobenzene	Methamoglobin < 1 % of Hb.
Chromium	Cr in blood < 0.1 micro gm / 100 ml. Cr. in urine < 0.05 micro gm / 100 ml
Arsenic	Should not be > 0.001mg/100ml of urine Sea food should be avoided for minimum two days.
Radiation	5 REM / year (50 m S v/ year)
Carbon disulphide	End of shift urine sample U-TTCA level not more than 5 mg/ gm of creatinine Iodine azide urine test can be used as a screening test
Mangenese	Blood level not more than 2 micro gm /100 ml Urine level not more than 2 micro gm / 100 ml. Faeces level not more than 60 mg per kg of faeces.
Dinitrophenol	presence of dinitrophenol or amino phenol in urine is diagnostic
Cadmium	Beta 2 micro globulin > 10 mg per litre in urine, diagnostic Cadmium metallothionein in urine >0.05 micro gm /100 ml is diagnostic.
Fluorine	level in urine pre shift < 0.4 mg / 100 ml post shift < 0.8 mg / 100 ml

Methanol	post shift urinary methanol should be less than 1mg/ 100 ml Absence of formic acid in urine does not rule out exposure.
2 propanol	Acetone in blood should be less than 5mg/100ml Acetone in urine should be less than 5mg/100ml.
Carbon monoxide	Carboxy Hb should not exceed 1% of Hb.
Acetonitrile	Cyanide in blood should not exceed 0.002mg/100 ml.
Ammonia	Blood urea Nitrogen 10-20 mg/100ml. Blood ammonia should not exceed 105microgm/100 ml Urinary urea nitrogen 4 gms per day.
Cotton Dust Byssinosis	Lung Function Test shows FEV_1 < 60% of predicted or FEV_1/FVC < 75%

Lists of Symptoms and causative agents

1

LUNG IRRITATION

Causative Agents

Acrolein
Ammonia
Antimony
ANTU
Beryllium and
beryllium compounds
Boron trifluoride
Bromine
Butyl mercaptan
Cadmium dust/fume
Chlorine
Chlorine dioxide
Chlorine trifluoride
1-Chloro-1-nitropropane
Chloropicrin
Chromic acid and
chromates
Chromium, metal and
insoluble salts
Cotton dust, raw
Diazomethane
Diborane
1,1-Dichloro-1-nitroethane
Dichloroethyl ether
Diisopropylamine
Dimethylamine
Dimethyl sulfate
Ethanolamine
Ethylene Chlorohydrin
Ethyleneimine
Ethylene Oxide
Ethyl mercaptan
Fluorine
Iodine
Maleic anhydride
Methyl bromide
Methylene biphenyl -
isocyanate
Methyl iodide
Methyl isocyanate
Methyl mercaptan
Nickel carbonyl
Nitric acid
Nitroethane
Nitrogen dioxide
2-Nitropropane
Oxygen difluoride
Ozone
Paraquat
Perchloromethyl mercaptan
Perchloryl fluoride
Phosgene
Phosphine
Phosphorus trichloride
Phthalic anhydride
Selenium hexafluoride
Silicon tetrafluoride
Sulfur dioxide
Sulphuric acid
Sulphur pentafluoride
Tellurium hexafluoride
Tolune -2, 4-diisocyanate
Tributyl phosphate
Uranium (natural), soluable
and insoluable compounds
Vanadium Pentoxide

Hydrogen chloride
Hydrogen fluoride
Hydrogen sulphide
Zinc chloride fume

2
ASPHYXIA
Causative Agents

Chemical Asphyxiants
Acetonitrile
Acrylonitrile
Carbon Monoxide
Cyanides (alkali)
Hydrogen cyanide

Simple Asphyxiants
Acetylene
Argon, neon and helium
Carbon dioxide
Dichloromonofluoromethane
Dichlorotetrafluoroethane
Ethane
Hydrogen
Liquified petroleum gas
Methane
Nitrogen

3
FIBROSIS
Causative Agents

Aluminium powder (stamped)
Asbestos
Coal dust
Cobalt, metal fume and dust
Hematite
Kaolin
Silica, amorphous including diatomaceous earth (when contaminated with crystalline silica)
Silica, crytalline
Yttrium

4
PNEUMOCONIOSIS
Causative Agents

Aluminium Powder
Mica

Barium and compunds
Cobalt, metal fume and dust
Graphite, natural
Hematite
Iron oxide fume
Kaolin
Silica, amorphous including natural diatomaceous earth
Soapstone
Stannic oxide
Talc, nonasbestos form

5

LUNG CANCER

Causative Agents

Arsenic and compounds
Asbestos
Bis (chloromethyl) ether
Chloromethyl methyl ether
Chromates
Coke oven emissions

6

CENTRAL NERVOUS SYSTEM DEPRESSION

Causative Agents

Acetaldehyde
Acetone
Acetylene dichloride
Allyl Glycidyl ether
n-Amyl acetate
sec-Amyl acetate
Benzen
Boromoform
1,3-Butadiene
n-Butyl Acetate
sec-Butyl Acetate
n-Butyl alcohol
sec-Butyl alcohol
tert-Butyl alcohol
n-Butyl glycidyl ether
Butyl mercaptan
Carbon disulfide
Carbon tetrachloride
Chlorobenzene
Isoamyl alcohol
Isobutyl acetate
Isobutyl alcohol
Isopropyl alcohol
Isopropyl ether
Mesityl oxide
Methyl acetate
Methyl acetylene
Methyl acetylene, propadiene mixture
Methyl amyl ketone
Methyl butyl ketone
Methyl cellosolve acetane
Methylcyclohexane
Methylcyclohexanol
o-Methylcyclohexanone
Methylene chloride
Methyl ethyl ketone
Methyl formate

Chlorobromomethane
Chloroform
Cresol, all isomers
Cumene
CyclohexaneCyclohexanol
Cyclohexanone
Cyclohexene
Decaborane
Diacetone alcohol
Dichlorodifluoromethane
Dichloroethyl ether
Difluorodibromomethane
Diglycidyl ether
Diisobutyl ketone
Dipropylene glycol
methyl ether
2-Ethoxyethyl acetate
Ethyl acetate
Ethyl alcohol
Ethul amyl ketone
Ethyl benzene
Ethyl bromide
Ethyl butyl ketone
Ethyl chloride
Ethylene dibromide
Ethylene dichloride
Ethylene oxide
Ethyl ether
Ethyl formate
Ethyl mercaptan
Ethylidene chloride
Furfuryl alcohol
Glycidol
n-Heptane
Hexachloroethane
n-Hexane
sec-Hexyl acetate
Isoamyl acetate
Methyl iodide
Methyl isobutyl carbinol
Methyl isobutyl ketone
Methyl mercapatan
Methyl propyl ketone
alpha-Methyl styrene
Naphtha, coal tar
Naphtha, petroleum distillates
Nitroethane
Octane
Pentaborane
Pentane
Phenyl glycidyl ether
Propyl acetate
n-Propyl alcohol
Propylene dichloride
Propylene oxide
Pyridine
Stoddard solvent
Styrene
Sulfuryl fluoride
1,1,1,2-Tetrachloro-2,difluorotha
1,1,2,2-Tetrachloro-1,2-
difluoroethane
Tetrachloroethane
Tetrachloroethylene
Tetrahydrofuran
Toluene
1,1,1-Trichloroethane
1,1,2-Trichloroethane
Trichloroethylene
Trochlorofluromethane
1,2,3-Trichlorofluromethane
1,1,2-Trichloro-1,2,2
trifluoroethane
Turpentine
Vinyltoluene
Xylene

7
CONVULSIONS
Causative Agents

Aldrin
2-Aminopyridine
Camphor
Chlordane
Crag Herbicide
DDT
Decaborane
2,4-Dichlorophenoxyacetic acid
Dieldrin
1,1-Dimethylhydrazine
Endrin
Heptachlor
Hydrazine
Lindane
Methoxychlor
Methyl bromide
Methyl chloride
Methyl iodide
Methyl mercaptan
Monomethylhydrazine
Nicotine
Nitromethane
Oxalic acid
Pentaborane
Phenol
Sodium Fluoroacetate
Tetraethyllead
Tetramethyllead
Tetramethylsuccinonitrile
Thallium, soluble compunds
Toxaphene

8
PERIPHERAL NEUROPATHY
Causative Agents

Acrylamide
Arsenic and compounds
Calcium arsenate
Carbon disulfide
n-Hexane
Lead and inorganic lead compunds
Lead arsenate
Mercury
Methyl bromide
Methyl butyl ketone
Thallium, soluble compunds
2,4,6-Trinitrotoluene

9

PRIMARY IRRITATION OF SKIN

Casuative Agents

AcetaldehydeAcetic acid
Acetic anhydride
Acetone
Acrolein
Acrylamide
Acrylonitrile
Allyl alcohol
Allyl glycidyl ether
Ammonia
n-Amyl acetate
sec-Amyl acetate
Antimony
Arsenic and compounds
Barium and compounds
Benzene
Benzoyl peroxide
Benzyl Chloride
Beryllium and Beryllium compounds
Boron oxide
Boron trifluoride
Bromine
n-Butyl acetate
sec-Butyl acetate
n-Butyl alcohol
sec-Butyl alcohol
n-Butylamine
n-Butyl Glycidyl ether
Calcium arsenate
Calcium oxide
Carbaryl
Carbon disulfide
Carbon tetrachloride
Chlorinated diphenyl oxide
Ketene
Lead arsenate
Lithium hydride
Maleic anhydride
Mercury
Mercury, alkyl compounds
Mesityl Oxide
Methyl acetate
Methyl acrylate
Methylal
Methyl alcohol
Methyl amine
Methyl bromide
Methyl butyl ketone
Methyl chloride
Methyl cyclo hexane
Methyl cyclo hexanol
o-Methylcyclohexanane
Methele bisphenyl isocyanate
Methylene chloride
Methyl ethyl ketone
Methyl iodide
Methyl isobutyl ketone
alpha-Methyl styrene
Monoethylhydrazine
Monomethylhydrazine
Morpholine
Naled
Naphtha, coal tar
Naphtha, petrollium distillates
Nickel, metal

Chlorine
Chlorine trifluoride
Chloraacetaldehyde
alpha-Chloroacetophenone
Chlorobenzene
o-Chlorobenzylidene malonitritle
Chlorobromomethane
Chlorodiphenyl, 42% chlorine
Chlorodiphenyl, 54% chlorine
Chloroform
Chloropircin
Chloroprene
Chromic acid and chromates
Chromium, soluble chromic and chromous salts
Coal tar pitch volatiles
Copper dusts and mists
Crag herbbicide
Cresol, all isomer
Crotonaldehyde
Cumene
Cyanides (alkali)
Cyclohexane
Cyclohexanol
Cyclohexanone
Cyclohexene
DDT
Diacetone alcohol
Dibutyl phosphate
o-Dichlorobenzene
p-Dichlorobenzene
1,1-Dichloro-1-nitroethane
Diethylamine
Diethylaminoethanol
Diglycidyl ether
Diisobutyl ketone
Nitric acid
Nitrobenzene
Nitroethene
Nitromethane
Octachloronaphthalene
Octane
Osmium Tetroxide
Oxalic acid
Pentaborane
Pentachloronaphtanlene
Pentacholorophenol
Pentane
Perchloromethyl mercaptan
Phenol
p-Phenylenediamine
Phenyl ether
Phenyl ether-biphenyl mixture
Phenyl glycidyl ether
Phenylhydrazine
Phosgene
Phosphoric acid
Phosphrous (yellow)
Phosphorus pentachloride
Phosporous pentasulfide
Phosporous trichloride
Phthalic anhydride
Picric acid
Platinum, soluble salts
Portland cement
Propyl acetate
n- Propyl alcohol
Propylene dichloride
Propylene imne
Propylene oxide
Pyrethrum
Pyridine
Quinone

Dimethylamine
Dimethylformamide
1,1-Dimethylhydrazine
Dimethyl sulfate
Dioxane
Epichlorhydrin
Epoxy resins
Ethanolamine
2-Ethoxy ethanol
2-Ethoxyethyl acetate
Ethyl acetate
Ethyl acrylate
Ethylamine
Ethyl amyl ketone
Ethylenediamine
Ethylene dibromide
Ethylene diclhloride
Ethyleneimine
Ethylene oxide
Ethyl ether
Ethyl formate
Ethylidene chloride
Ethyl silicate
Fluoride
Fluorine
Formaldehyde
Formic acid
Furfural
Glycidol
n-Heptane
Hexachloronaphthalene
n-Hexane
Hydrazine
Hydrogen bromide
Hydrogen chloride
Hydrogen fluoride
Hydrogen peroxide, 90%
Iodine
Isomyl acetate
Rotenone
Selenium compounds
Silver, metal and
soluble compounds
Sodium hydroxide
Stoddard solvent
Styrene
Sulfur dioxide
Sulfur monochloride
Sulphuric acid
Tellurium
Terphenyls
Tetrachloroethane
Tetrachloroethylene
Tetrachloronaphthalene
Tetranitromethane
Tetryl
Thiram
Tin, organic and
inorganic compounds
Tolene
Toluene 2,4-diisocyanate
o-Toluidine
Toxaphene
Tributyl phosphate
1,1,1-Trichloro ethane
Trichloro ethylene
Trichloro naphthalene
2,4,5-Trichloro phenoxy
acetic acid
1,2,3-Trichloro propane
1,1,2-Trichloro-1,2,2-Tri-
fluoro ethane
Tri ethylamine
2,4,6-Trinitro toluene
Turpentine
Uranium (natural), soluble
and insoluble compounds.
Van adium pentoxide

Isobutyl alcohol
Isophorone
Isopropylamine
Isopropyl ether
Isopropyl glycidyl ether
Vinyl toluene
Xylene
Zinc chloride fume

10
SENSITISATION OF SKIN
Casuative Agents

Acetaldehyde
Acetic acid (rare)
Acetic anhydride
Allyl glycidyl ether
Arsenic and compunds
Benzoyl peroxide
Beryllium and beryllium compounds
n-Butyl glycidyl ether
Chromic acid and chromates
Cobalt, metal fume and dust
Copper dusts and mists
Cresol, all isomers
o-Dichloro benzene
Epoxy resins
Ethyl acrylate
Ethylenediamine
Ferbam
Formaldehyde
Formic acid
Iodine
Isopropyl glycidyl ether
Maleic anhydride
Mercury
Naphthalene
Nickel, metal
Nito benzene
p-Phenylenediamine
Phenyl glycidyl ether
Phenylhydrazine
Phthalic anhydride
Picric acid
Platinum, soluble salts
Pyrethrum
Selenium compounds
Tetryl
Thiram
Toluene2,4-diisocyanate
2,4,6-Trinitro toluene
Vanadium pentoxide

11
DAMAGE TO LIVER
Causative Agents

Acetylene tetrabromide
Kepone

Carbon dissulfide
Carbon tetrachloride
Chlorodiphenyl
Chloroform
p-Dichlorobenzene
Dimethyl acetamide
Dioxane
Ethylene chlorohydrin
Ethylene dibromide
Ethylene dichloride
Hexa chloro naphthalene
Nitroethane
Octa chloro naphthalene
Pentachloronaphthalene
Picric acid
Tetrachloroethane
Tetrachloethylene
Tetracholoronaphthalene
Tetryl
Trichloronaphthalene
2,4,6-Trinitrotoluene

12
RENAL PROBLEMS
Casuative Agents

4-Aminodiphenyl Arsine
Butyl cellosolve
Benzene
Carbon disulfide
Carbon tetrachloride
Choloroform
Dinitrophenol
Dioxane
Ethylene chlorohydrin
Ethylene dibromide
Mercury
Naphthalene
Oxalic acid
Phenylhydrazine
Picric acid
Stibine
Tetryl
Tetrachloroethane
2,4,6-Trinitrotoluene
Turpentine
Uranium (natural),
soluble and
insoluble compounds

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